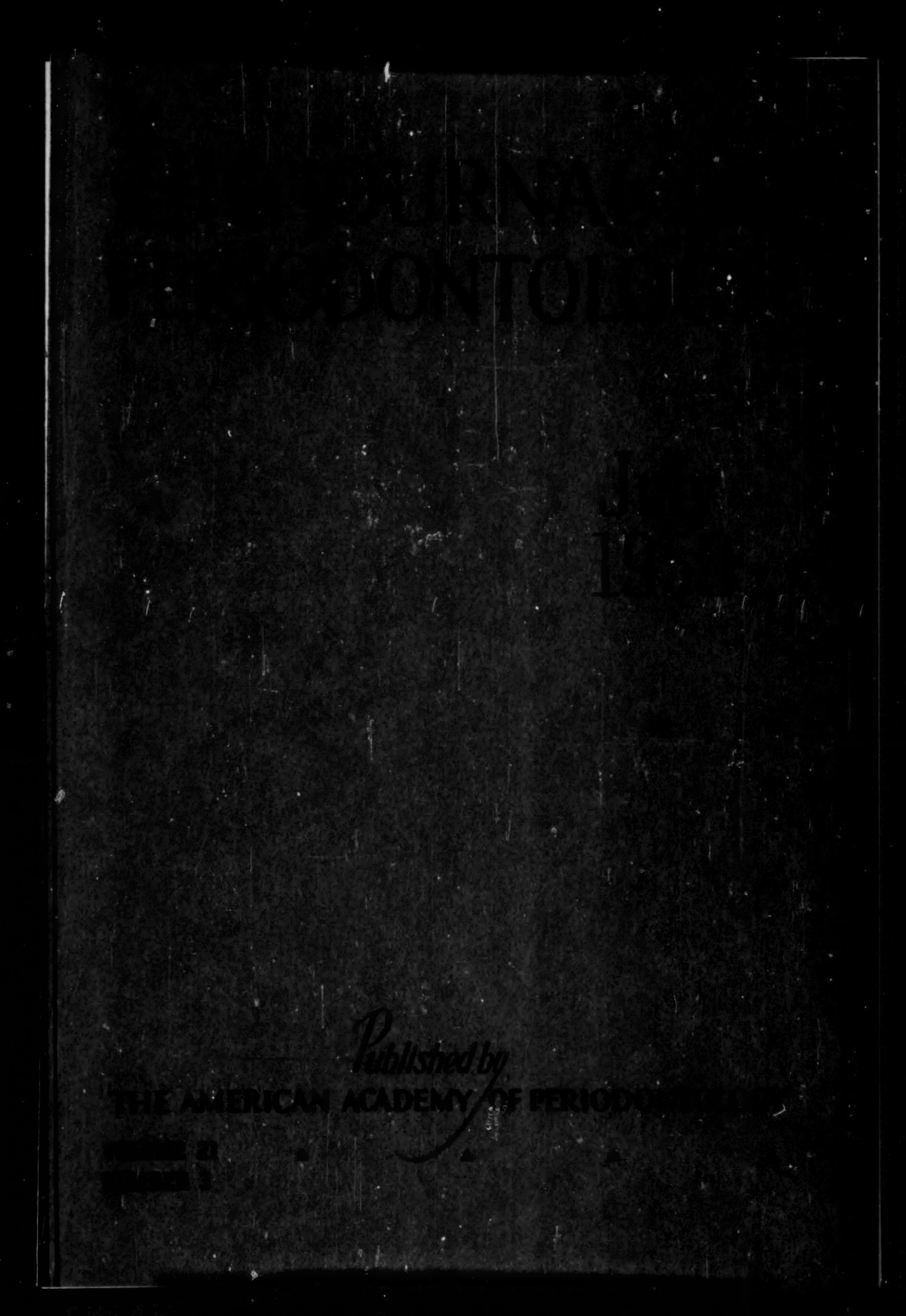
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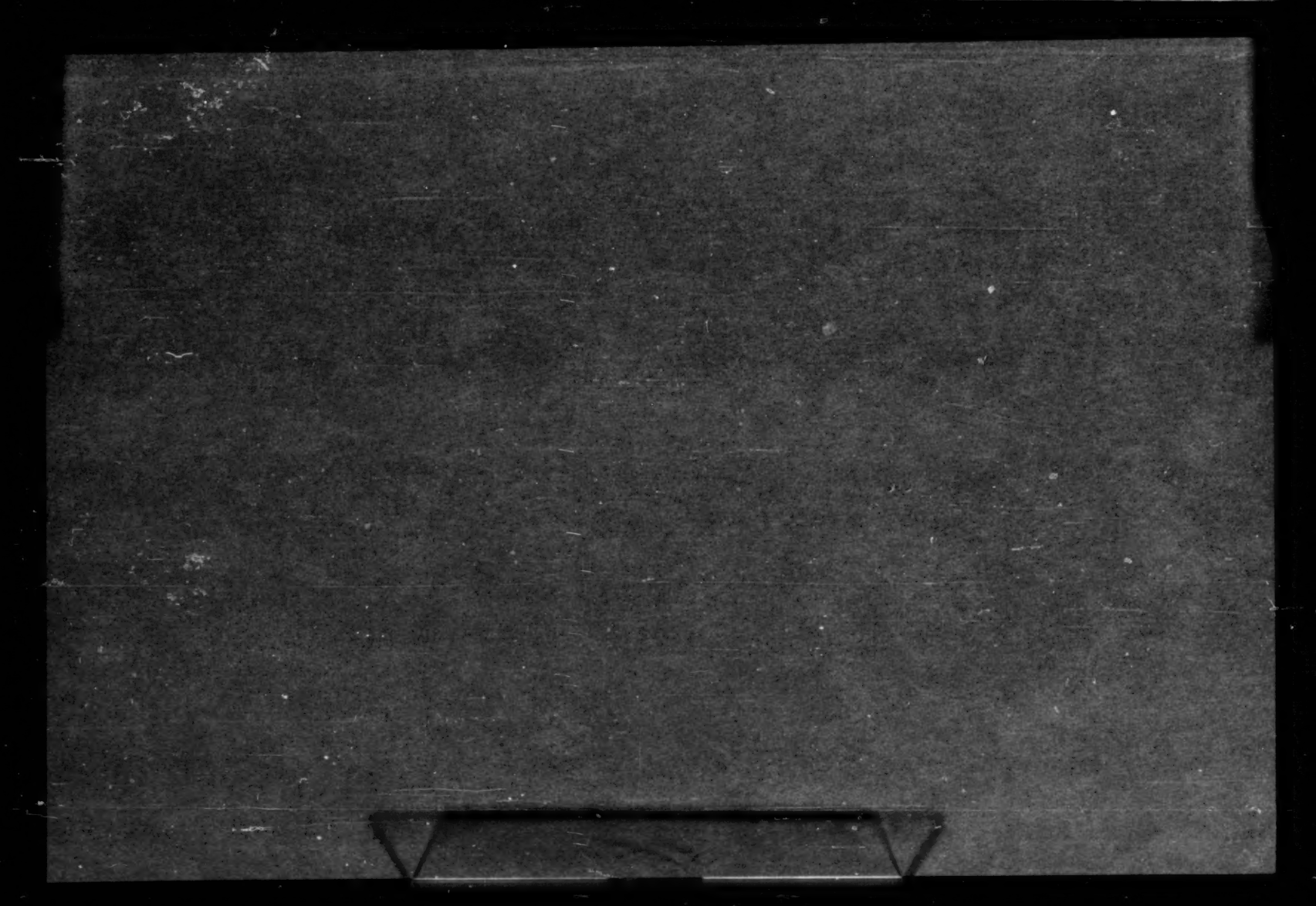

The Journal of Periodontology
is dedicated to the memory of
Dr. Gillette Hayden
for her pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
of Periodontology

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

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Thirty-fourth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

October 26, 27, and 28, 1950

Atlantic City, N. J.

Bernhard Gottlieb, M.D., LL.D., D.M.D.

1885-1950

Dentists everywhere were saddened to learn of the death of Dr. Bernhard Gottlieb on March 15, 1950. His research activities had made him one of this century's outstanding dental scientists.

Dr. Gottlieb was born in Kuty, Poland in 1885, and held the degree of M.D. of Vienna (1911) and the honorary degrees of LL.D., Loyola, Chicago and D.M.D. of Bonn. From 1911 until 1938 he taught at the University of Vienna; during this time he developed the research laboratory which attracted world-wide attention and to which dental investigations came from many countries.

Chaotic political conditions forced him to leave Vienna in 1938, and after a period of intense activity devoted to the building up of the dental profession in Palestine, he came to America. Attached first to the University of Michigan, he later became Professor and Head of the Department of Pathology and Dental Research of the College of Dentistry, Baylor University, Dallas, Texas. He carried on there until his death on March 15, 1950.

Dr. Gottlieb married in 1917, and his widow, Stella, passed away a few weeks after his death. A son, Erich, is now engaged in postgraduate study in Columbia University, looking toward a career in political science.

Dr. Gottlieb earned the respect and gratitude of the dental professions everywhere for his many contributions to dental science. He was particularly interested in periodontology and was an Honorary Member of the American Academy of Periodontology.

R.G.A.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FIFTY

A Basic Classification of "Gingival Enlargement"

IRVING GLICKMAN, B.S., D.M.D.

INCREASE in the size of the gingiva is a common clinical feature of gingival disease. Common usage has grouped all gingival enlargements other than tumor masses under the all-inclusive term "hypertrophic gingivitis" purely on the basis of the clinical appearance of increased size and without regard for the nature of underlying histopathological changes. Because it has been used to connote clinical conditions which are fundamentally different, the term "hypertrophic gingivitis" has been of limited diagnostic value. It has also confused the problem of the treatment of gingival disease by obscuring the nature of the many conditions it ostensibly describes. A need exists for the clarification of the nature of gingival enlargements in order to provide a rational basis for diagnosis and treatment.

The initial step in the clarification of gingival enlargement entails elimination of the term "hypertrophic gingivitis." Hypertrophy connotes "increase in the size of an organ as a result of increase in size of its individual component cells with a view of meeting increased functional requirements for useful work."[1] Enlargement of the gingiva in gingival disease is neither primarily the result of an increase in size of component cells, nor does it generally occur in response to increased functional requirement for useful work. The inadequacy of the term "hypertrophic gingivitis" stems from its basic inaccuracy.

The treatment of gingival disease should be preceded by intelligent observation and comprehensive diagnosis. For the purpose of describing the gingival conditions in which increase in size is the prominent clinical feature—a non-specific descriptive term should be used. The term "gingival enlargement"[2] is such a term. It merely describes the clinical finding. It does not attempt an explanation of its fundamental nature. The latter information evolves from a thorough analysis of individual cases. When a diagnosis is reached, the term "gingival enlargement" is then appropriately qualified. The differentiation between observation and diagnosis is a prerequisite preliminary to rational treatment.

To enhance the effectiveness of the term "gingival enlargement" it should include reference to the location and distribution of the condition under consideration. This is accomplished by use of the following terms:

"Localized" —Limited to the gingiva in relation to a single tooth or group of teeth.

"Generalized"—Involving the gingiva throughout the mouth.

"Marginal" —Involvement limited to the gingival margin.

"Diffuse" —Involving the gingival margin and the remainder of the gingival mucosa up to the mucobuccal fold and for a varying distance along the lingual surface.

An individual case may, therefore, be described as a localized marginal gingival

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enlargement, localized diffuse gingival enlargement, generalized marginal gingival enlargement, or generalized diffuse gingival enlargement. An isolated tumor-like lesion is described as a discrete gingival enlargement.

For diagnostic purposes gingival enlargement may be classified according to underlying histopathological changes and etiology as follows:

I. INFLAMMATORY
- A. Chronic Inflammatory Gingival Enlargement
  - 1. Generalized or localized
  - 2. Discrete (Tumor-like)
- B. Acute Inflammatory Gingival Enlargement (Gingival abscess)

II. NON-INFLAMMATORY HYPERPLASTIC GINGIVAL ENLARGEMENT (Gingival hyperplasia)
- A. Marginal
- B. Diffuse

III. COMBINED GINGIVAL ENLARGEMENT

IV. CONDITIONED GINGIVAL ENLARGEMENT
- A. Hormonal
  - 1. Gingival Enlargement of Pregnancy
  - 2. Gingival Enlargement of Puberty
- B. Leukemic Gingival Enlargement
- C. Gingival Enlargement Associated with Vitamin C Deficiency

V. NEOPLASMS

VI. DEVELOPMENTAL GINGIVAL ENLARGEMENT

I. INFLAMMATORY

Gingival enlargement may result from chronic or acute inflammatory changes. The former is by far the more common.

A. CHRONIC INFLAMMATORY GINGIVAL ENLARGEMENT

Clinical Features

1. Generalized or localized—

Chronic inflammatory gingival enlargement may involve the gingiva in relation to a single tooth or the gingival margin throughout the mouth. It may therefore be described as either localized or generalized. It is usually marginal in location and can be distinguished from the adjacent uninvolved gingival mucosa on the basis of its appearance. It originates as a slight labial and lingual ballooning of the marginal gingiva with an associated bulging of the interdental gingival papilla. In the early stages the effect is the production of a "life saver" like exaggerated gingival contour around the necks of the involved teeth. This increases in size until it appears as a distended semi-spherical extension of the adjacent uninvolved mucosa protruding labially, lingually, and interproximally and covering portions of the enamel of the involved teeth. In general, such chronic inflammatory increase in size progresses slowly and painlessly unless it is complicated by secondary bacterial or traumatic involvement. In most instances there is a tendency toward hemorrhage in response to slight surface irritation.

2. Discrete (Tumor-like)—

Less frequently chronic inflammatory gingival enlargement takes the form of a discrete sessile or pedunculated mass which protrudes from the interproximal papilla or the labial or lingual gingival margin. These "tumor-like" inflammatory lesions are slowly growing and painless and may even undergo spontaneous reduction in size only to reappear and enlarge as time progresses.

Underlying Microscopic Features—

The following microscopic changes are responsible for chronic inflammatory gingival enlargement and its associated clinical alterations:

1. Fluid and cellular exudate
2. New capillary formation
3. Vascular engorgement
4. Hemorrhage
5. Proliferation of connective tissue and epithelium associated with inflammation
6. Degeneration of connective tissue and epithelium

Chronic inflammatory gingival enlargement is characterized by changes in color, consistency and texture which are governed by variations in the above histological components. Lesions which consist of a predominance of inflamma-

tory cells and fluid with associated degenerative changes are deep red or bluish red in color and soft and friable with a smooth shiny surface. Lesions which are predominantly fibrotic with an abundance of fibrocytes, fibroblasts and bundles of collagen are relatively firm, resilient and pink.

Any local condition capable of producing chronic inflammation may result in gingival enlargement. Included amongst a multitude of sources of irritation are calculus, overhanging fillings, ill-fitting crowns, occlusal trauma, poor hygiene, lack of function, carious lesions, improper prosthesis, and "food impaction" with all of the local alterations which produce it.

B. ACUTE INFLAMMATORY GINGIVAL ENLARGEMENT

(Gingival Abscess)

Clinical Features—

This is a localized, painful, rapidly expanding lesion which appears rather suddenly. It is generally limited to the marginal gingiva or interdental papilla. In its early stages it appears as an erythematous mound-like swelling with a smooth, shiny surface. Within 24 to 48 hours, the lesion is usually fluctuant and pointed with a superficial orifice from which a purpulent exudate may be expressed. There is frequently an accompanying pericementitis of the contiguous tooth or teeth. If permitted to progress, the lesions generally rupture spontaneously.

Underlying Microscopic Features—

The lesions generally consist of a purulent focus in the connective tissue and an associated diffuse infiltration of polymorphonuclear leukocytes, edema and vascular engorgement. The overlying epithelium presents varying degrees of intra and extra cellular edema, invasion by leukocytes with ulceration.

Acute inflammatory gingival enlargement constitutes a response to irritation produced by foreign substances such as toothbrush bristle, apple core, or lobster shell forcefully embedded into the gingival sulcus.

II. NON-INFLAMMATORY HYPERPLASTIC GINGIVAL ENLARGEMENT (Gingival Hyperplasia)

By definition "hyperplasia" refers to an increase in the size of a tissue or organ produced by an increase in the number of its component cells. There are conditions in which gingival enlargement results from an increase in the number of its component cells unrelated to inflammation. Such enlargement is in reality a gingival hyperplasia. An important clinical feature of gingival hyperplasia is that in its uncomplicated state increase in size and change in shape are its only abnormal characteristics. The gingiva is pale pink, firm and resilient and presents none of the changes in color, surface texture or consistency or tendency toward hemorrhage generally associated with inflammatory gingival enlargement.

Clinical Features—

Non-Inflammatory Hyperplastic Gingival Enlargement occurs in two forms: a) marginal; and b) diffuse.

a) Marginal non-inflammatory hyperplastic gingival enlargement appears initially as mulberry-like minutely lobulated protuberences which seem to emerge from beneath the free gingival margin. These protuberances can generally be separated from the apparently uninvolved marginal gingiva by a linear fissure into which a probe may be inserted for a slight depth. This is an additional clinical feature which serves to distinguish this condition from inflammatory enlargement, the surface of which is continuous with the adjacent gingival mucosa. The hyperplastic protuberances occur labially, lingually, and interproximally and enlarge until they constitute a curtain-like projection over varying portions of the enamel. Involvement of the gingiva may be limited to a small area in relation to a group of teeth or may be generalized throughout the mouth.

Marginal gingival hyperplasia is the type of gingival enlargement associated with the use of Dilantin Sodium.[3-6]

b) Diffuse Non-Inflammatory Gingival Enlargement: In this condition, the entire gingival mucosa up to the mucobuccal fold and including the gingival margin is enlarged. Both labial and lingual surfaces are involved. The overall effect is an apparent distortion in the contour of both the mandible and maxilla. Isolated cases involving either the maxilla or mandible have been

reported. The gingiva is pink, almost leathery in consistency and presents a characteristic minutely "pebbled" surface. This type of gingival enlargement is comparatively uncommon. It has been described by such terms as "gingivomatosis" or "elephantiasis"[7] "diffuse fibroma"[8] "familial elephantiasis"[9] "idiopathic fibromatosis."[10]

Underlying Microscopic Features—

The epithelium for the most part is thickened and presents marked acanthosis and extension of tortuous elongated rete pegs for a considerable depth into the underlying connective tissue. The connective tissue consists of densely arranged collagen bundles with an increase in adult and young connective tissue cells.

The etiology of diffuse gingival hyperplasia remains to be clarified. Familial tendency, neoplasm,[11] nutritional and endocrinological disturbances[12] have been suggested as causative factors.

Distinction should be made between the bulbous distortion in the surface contour of the mandible and maxilla produced by diffuse gingival hyperplasia and the actual distortion in the shape of these bones associated with marked malocclusion. In the latter condition the gingiva may be essentially unaltered or may present no pathological changes other than chronic marginal inflammation in relation to the malposed teeth. However, the combined appearance of the marginal gingiva and the unaltered mucosa covering the surface of the deformed bone may lead to the erroneous impression of diffuse gingival enlargement. Intelligent diagnosis will avoid extensive surgical procedures in many instances in which judicious care of the marginal condition will accomplish all that may reasonably be expected from periodontal treatment.

III. COMBINED GINGIVAL ENLARGEMENT—

This condition results when gingival hyperplasia is complicated by secondary inflammatory changes. Gingival hyperplasia provides conditions favorable for the accumulation of debris by accentuating the depth of the gingival sulcus, by interfering with effective hygiene measures and by deflecting the normal excursive pathways of food in the course of mastication. The secondary inflammatory changes thus produced accentuate the size of the pre-existent gingival hyperplasia.

Alterations in the clinical features of the gingiva other than increased bulk are also produced by the introduction of inflammation. These include a change from the previous pink color to various shades of red and bluish-red, change from minutely lobulated or pebbly appearance to a ballooned out distended shiny surface, and change from a firm consistency to varying degrees of softness and friability. When the secondary inflammation is slight and limited to the margin, it is possible to clinically appraise the real nature of the condition because the features of the pre-existent non-inflammatory enlargement predominate. However, in many instances the extent of secondary inflammation is such as to obscure the features of the pre-existent non-inflammatory involvement and create the typical appearance of chronic inflammatory enlargement. The latter condition is far less frequent than simple chronic inflammatory gingival enlargement, but more common than uncomplicated non-inflammatory gingival hyperplasia.

It is essential to the satisfactory clinical management of gingival enlargements in general that the real nature of combined gingival enlargement be understood. It consists of two components, a primary or basic hyperplasia of connective tissue and epithelium, the origin of which is unrelated to inflammation, and a secondary complicating inflammatory component.

This analysis may be represented as follows:

The removal of all sources of local irritation in cases of combined gingival enlargement (Fig. 3) will eliminate the secondary inflammatory component. The overall size of the lesion will be reduced in accordance with the degree to which inflammation is a secondary contributing feature. The non-inflammatory hyperplasia will remain. Elimination of the non-inflammatory hyperplasia requires further exploration of the causative factors and their correction when possible.

This clarification of the nature of combined gingival enlargement is particularly pertinent in the periodontal treatment of patients receiving Dilantin Sodium. The gingival enlargement in such cases may be of three types:

Type I.—Inflammatory Enlargement entirely unrelated to the Dilantin therapy. (Fig. 1)

FIGURE 1

INFLAMMATORY ENLARGEMENT

FIGURE 2

NON-INFLAMMATORY HYPERPLASTIC ENLARGEMENT

FIGURE 3

COMBINED ENLARGEMENT

Type II.—Non-Inflammatory Hyperplastic Enlargement associated with the Dilantin (Fig. 2)

Type III.—Combined Enlargement (Fig. 3)

In accordance with the above evaluation one of the following three responses to local periodontal treatment is possible in patients under Dilantin therapy:

In Type I cases—Complete elimination of the enlargement.

In Type II cases—No lasting effect upon the enlargement.

In Type III cases—Reduction in size of the enlargement depending upon the degree of secondary inflammatory complication.

Regardless of the method of local treatment, complete elimination of the Type II and Type III enlargement will not occur unless Dilantin therapy is discontinued. If this is done, no local treatment is required in the Type II cases.

IV. CONDITIONED GINGIVAL ENLARGEMENT

This type of enlargement occurs when the systemic condition of the patient is such as to exaggerate or distort the usual gingival response to local irritation. Alteration in the usual microscopic inflammatory response produces a corresponding modification of the usual clinical features of chronic inflammatory gingival enlargement. The specific manner in which the clinical picture of conditioned gingival enlargement differs from chronic inflammatory enlargement depends upon the nature of the systemic modifying influence. Local irritation is necessary for the initiation of this type of enlargement. It does not, however, solely determine the nature of its clinical features.

The following are the types of conditioned gingival enlargement (A-C):

A. HORMONAL

1. Gingival Enlargement in Pregnancy

Clinical Features—

In pregnancy, gingival enlargement may appear as a generalized bulbous increase in the size of the marginal gingiva or as simple or multiple discrete tumor-like masses.

(a) Marginal Gingival Enlargement—

The prevalence of marginal gingival enlargement in pregnancy has been reported as 10%[13] and 70%.[14] Marginal gingival enlargement in pregnancy may result from the accentuation of a previously enlarged area or may appear in areas which presented no notable previous clinical changes. Although macroscopically unaltered, some degree of gingival inflammation does exist in the latter instances. Marginal gingival enlargement of pregnancy does not occur in areas entirely devoid of irritation.

The clinical picture varies considerably. The enlargement tends to be more prominent interproximally than on the labial and lingual surfaces. In some cases the enlarged gingiva is bright red, soft and friable. In others it is dusky blue or magenta in color with a smooth shiny surface. In all instances

the enlargement can be clearly differentiated from the adjacent mucosa. Bleeding may be elicited upon the slightest surface stimulation.

In patients with some degree of pre-existent marginal enlargements, it is frequently possible to observe clinical changes as pregnancy progresses. Although such uncontrolled clinical observations cannot be interpreted with any degree of conclusiveness, it is apparent to some cases of pregnancy that pre-existent marginal inflammation becomes considerably enlarged, the color assumes a magenta shade, the surface smoothness is accentuated, the gingiva becomes soft and friable and the tendency to gingival bleeding increases.

Although pregnancy does not affect the gingiva so as to initiate gingival enlargement it frequently does result in notable enlargement in areas of pre-existent inflammation.

(b) Discrete Gingival Enlargement of Pregnancy—

Clinical Features—

The so-called "pregnancy tumor" is not a neoplasm. It is an inflammatory lesion of the gingiva which differs in clinical appearance from the usual inflammatory enlargement because it is the result of a microscopic response to local irritating factors which has been modified by the condition of the patient. It usually appears after the 3rd month of pregnancy with a reported incidence of 1.8%[14] and .5%[15] It is usually a painless lesion unless its size and shape are such as to foster accumulation of debris under its margin or interfere with the occlusion in which cases painful inflammatory changes may occur.

It appears as a discrete mushroom like flattened spherical mass with indentations on its lobulated periphery. It protrudes from the gingival margin in the interproximal (most common), labial or lingual areas to which it is attached by a sessile or pedunculated base. It tends to expand laterally while pressure from the tongue and cheek retain its flattened appearance. It is a superficial lesion which does not invade the underlying bone. It is generally dusky red or magenta in color with a smooth glistening surface which frequently presents numerous deep red pin-point markings. Localized erythema or grey necrosis occasionally modify the appearance at the periphery.

The consistency of the lesions varies from its usual semifirmness to present varying degrees of softness and friability.

Microscopic Features—

In both the marginal and discrete enlargement the gingiva consists of a circumscribed mass of connective tissue, the periphery of which is outlined by stratified squamous epithelium. The connective tissue consists for the most part of numerous diffusely arranged newly formed engorged capillaries lined by cuboidal endothelial cells. Between the capillaries there is a moderately fibrous stroma which presents varying degrees of edema and leukocytic infiltration. The peripheral stratified squamous epithelium is for the most part thickened with prominent rete pegs. The basal epithelium presents some degree of intra and extra-cellular edema with prominent intercellular bridges and infiltration by leukocytes. The surface of the epithelium is generally keratinized.

Invariably, these lesions present a zone of pronounced inflammatory involvement in which lymphocytes and plasma cells predominate amidst a scattering of polymorphonuclear leukocytes. The acute inflammatory zones generally occur immediately beneath the surface in relation to the margin.

The true nature of this lesion is indicated by the microscopic findings. The prominent endothelial proliferation with capillary formation and associated inflammation constitute its characteristic features. This pronounced capillary formation constitutes the modification of the usual gingival response to chronic irritation and is responsible for the unique clinical lesion. On the basis of the microscopic findings, gingival enlargement in pregnancy may be considered an angiogranuloma. This term is consistent with the basic inflammatory nature of the lesion and avoids the neoplastic implications of such terms as fibro-hemangioma or pregnancy tumor. Maier and Orban[15] point to the proliferative tendencies in the epithelium, capillary endothelium and connective tissue as the most characteristic features of the gingivae in pregnancy, but add that they are not pathognomonic of pregnancy, nor can they be used as a

reliable basis for differentiating between gingivitis in pregnant and non-pregnant individuals.

The elimination of all sources of local irritation will minimize the likelihood of gingival enlargement in pregnancy. The treatment of gingival enlargement of pregnancy which is limited to the removal of tissue and disregards local irritation will be followed by recurrence.

Although spontaneous reduction in the size of gingival enlargements commonly follows the termination of pregnancy, the complete elimination of the residual inflammatory lesions is a prerequisite for intelligent periodontal care.

2. Gingival Enlargement of Puberty

Clinical Features—

Enlargement of the gingiva is frequently seen during puberty. It occurs in both males and females, and appears in areas of local irritation. The size of the gingival enlargement is, however, far in excess of that which usually is seen associated with comparable local factors. It is marginal in distribution and characterized by particularly prominent bulbous interproximal papillae. Frequently only the labial or buccal gingivae are enlarged, the lingual surfaces appearing unaltered.

In addition to increase in size, gingival enlargement of puberty presents all of the clinical features generally associated with chronic inflammatory gingival disease. It is the degree of enlargement and tendency toward massive recurrence in the presence of the slightest local irritation which differentiates the gingival enlargement of puberty from the uncomplicated increase in size associated with chronic inflammation. With the advent of young adulthood, spontaneous reduction in size and the cessation of post-treatment recurrences are frequently sequellae in cases of gingival enlargement of puberty.

Microscopic Features—

Because this condition is predominantly inflammatory in nature it is difficult to discern the conditioning influence of the systemic status of the patient in terms of specific histological changes. The microscopic picture is that of chronic inflammation with prominent edema and associated degenerative changes.

B. LEUKEMIC GINGIVAL ENLARGEMENT

Clinical Features—

In leukemic patients it is necessary to distinguish between true leukemic gingival enlargement and increase in the size of the gingiva produced by uncomplicated chronic inflammation. True leukemic enlargement occurs when, in response to local irritation, the gingiva is densely infiltrated with immature and proliferating leukocytes. The clinical picture presents an associated distortion. In contrast with true leukemic enlargement is the uncomplicated chronic inflammatory increase in the size of the gingiva which occurs in leukemic patients. In the latter condition the inflammatory response is entirely comparable to that seen in non-leukemic individuals. True leukemic enlargement seldom occurs in chronic leukemia. It occurs in acute leukemia in the presence of gingival irritation.

True leukemic enlargement is usually marginal and may be localized or generalized. It may appear as an oversized extension of the marginal gingiva, or as a discrete tumor-like mass attached to the gingival mucosa. Discrete enlargement may be single or multiple, irregularly spherical in shape and painless unless complicated by secondary inflammation. True leukemic enlargement is generally a dusky bluish-red in color with an irregularly indented shiny surface. The consistency is moderately firm with a tendency toward friability and hemorrhage. Acute ulcero-membranous involvement frequently occurs in the crevice formed by the junction of the enlarged gingiva and the contiguous mucosa.

Microscopic Features—

In true leukemic gingival enlargement the connective tissue is diffusely infiltrated with a dense mass of immature and proliferating leukocytes, the specific nature of which varies with the type of leukemia. Mature leukocytes associated with chronic inflammation are also seen. The capillaries are engorged. The connective tissue is, for the most part, edematous and degenerated. The epithelium presents varying degrees of leukocytic infiltration with edema. Isolated surface areas of acute necrotizing inflammation with a pseudomembranous fibrinous meshwork of fibrin, necrotic epithelial cells, polymorphonuclear leukocytes and bacteria are frequently associated microscopic findings.

C. GINGIVAL ENLARGEMENT ASSOCIATED WITH VITAMIN C DEFICIENCY

Clinical Features—

Enlargement of the gingiva is generally included in classical descriptions of the oral cavity in scurvy. It is important to recognize that such enlargement is essentially a conditioned response to local irritation. Acute Vitamin C deficiency does not of itself cause gingival inflammation.[16] It does result in hemorrhage, collagen degeneration, and edema of the gingival connective tissue. These changes modify the response of the gingiva to local irritation so that the normal defensive delimiting reaction is inhibited and the extension of the inflammation exaggerated.[17] The combined effect of acute Vitamin C deficiency and inflammation produce the massive gingival enlargement of scurvy.

Gingival enlargement associated with Vitamin C deficiency is marginal in distribution, bluish-red in color, soft, friable, hemorrhagic with a smooth shiny surface. It occurs associated with local irritation. Superficial necrosis with pseudomembrane formation is often a complicating feature.

Microscopic Features—

The gingiva presents a chronic inflammatory cellular infiltration with a superficial acute response. There are scattered areas of hemorrhage with engorged capillaries. Marked diffuse edema and collagen degeneration and absence of new collagen fibrils of fibroblasts are striking microscopic findings.

V. NEOPLASMS

Tumors of the gingiva constitute another aspect of the problem of gingival enlargement. The inclusion of "Neoplasms" as a generic term in this overall classification of gingival enlargements, rather than the presentation of a detailed description of different types of tumors of the gingiva will serve the requirements of this paper.

VI. DEVELOPMENTAL GINGIVAL ENLARGEMENT

Clinical Features—

This type of enlargement occurs in the course of growth and development of the jaws and is seen in the period of mixed dentition. It appears as a bulbous distortion of the labial and marginal contours of the gingiva in relation to teeth which are in various stages of emergence to the line of occlusion. Quite frequently the enlarged gingival contour persists in early adulthood until the epithelial attachment has migrated from the enamel surface to the cementoenamel junction.

In its strictest sense, developmental gingival enlargement may be construed as physiological in nature. It is, however, often complicated by marginal inflammatory changes. The composite picture thus produced lends itself to a superficial impression of extensive gingival enlargement. In reality, the condition is that of a marginal pathological change which is generally easily corrected superimposed upon a developmental malformation which will be appropriately modified in the course of time.

Microscopic Features—

When uncomplicated by inflammation the gingival tissues present no notable pathological changes. A zone of chronic inflammation at the gingival margin, is however, a common finding.

RECAPITULATION

Increase in the size of the gingiva is a non-specific clinical manifestation which may result from different types of underlying pathological changes. The term "hypertrophic gingivitis" is inaccurate and inconsistent with the nature of the different conditions it purports to describe. "Gingival enlargement" is preferred as a non-specific term for the description of conditions in which the gingiva is increased in size.

A classification of "gingival enlargement" based upon underlying histopathological changes has been offered. It is suggested that such classification eliminates the confusion created by the indiscriminate consideration of "hypertrophic gingivitis" as a pathological entity and thereby affords a rational basis for the diagnosis and treatment of conditions in which the gingiva is increased in size.

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The Rationale of Pocket Elimination in the Treatment of Periodontal Disease

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THE INCOMPLETE knowledge of the etiology of the diseases of the periodontium precludes the total management of these common oral disturbances. Dental science still remains very much "in the dark" as to their basic causes, and it follows that the successful prevention of periodontal disease is still beyond the realm of practical accomplishment. Following their inception, however, it is often possible to check their progressive course and bring them under a certain measure of control. This paradoxical situation is largely possible because of effective means of removing the symptoms of these pathological conditions. These measures enable the supporting tissues of the teeth to be freed of infective processes with their subsequent restoration to a state of health. In turn, the longevity and function of the teeth may be prolonged with proper care, over periods of infinite duration.

It may be stated that the current position regarding periodontal disease is relatively similar to that of dental caries. The goal of prevention in both oral problems remains to be attained, and the most practical approach to them, for the present, lies in means of control. Until such time as we possess a broader knowledge of their causes, we must resort for the major part of their correction to the treatment of their symptoms. The complete removal of these alone does not, however, alter those factors that have brought about their inception. Progress in their prevention must perforce be closely associated with advancement in the knowledge of their etiology. The advantages that may be gained by the removal of their symptoms are: the elimination of those pathological factors conducive to their further development and the return of the diseased tissues to a sound and healthy condition. The symp-

toms of both caries and periodontal disturbances, regardless of our eventual knowledge of their causes, will always require elimination, owing to their self-destructive nature and the inability of the affected tissues to restore themselves.

Whatever knowledge we may possess of both local and systemic factors that may influence the predisposition to or inception of periodontal diseases, should constitute an integral part of periodontal treatment. The physical condition of the patient should always be carefully considered with the cooperation, whenever possible, of the family physician. We cannot overlook the fact that metabolic and gastro-intestinal disturbances, vitamin, mineral and endocrine deficiencies, blood dyscrasias and any debilitating diseases all have a close relationship with the resistance and health of the periodontal tissues. The local factors are largely mechanical in nature and generally result in irritation followed by inflammation and infection. Their correction, either by removal, such as that of calculus and overhanging margins of restorations and crowns, or by alteration, such as grinding to reshape incisal or occlusal anatomy to relieve stresses and trauma, should always be carried out.

In the symptomatic treatment of periodontal disease the one point upon which many periodontists seem to agree, at least in principle if not always in practice, deals with the elimination of the pocket or lesion as being the prime objective. Coolidge,[1] among others, believes that the purpose of treatment of suppurating pockets is to check the suppuration and eliminate the pocket in which it exists. He further believes that its objective is to check the disease and prevent its further spread to deeper tissues, in order that the teeth may remain in function as long as possible without any harmful influence on general health or the local structures. Such a result might be considered the optimum in present-day periodontal therapy.

When we arrive at a better understanding of principles in biology it may then become possible to cause regeneration of the bone as well as that of cementum, and reintegrate a normal union and relationship between these two tissues. For the present there has been no basis established, either on clinical grounds or from microscopic evidence, to prove that any form of treatment can bring about the closing of a periodontal pocket through means of a connective tissue fibre attachment, from the detached soft tissue wall to the opposing necrotic cementum.

DEFINITION OF POCKET

In broad terms a periodontal pocket or lesion may be defined as a gingival crevice or sulcus of abnormal depth. Kronfeld[2] has stated that the depth of the normal gingival crevice varies from zero to several millimetres, and that the majority of crevices around human teeth measure between 0.1 and 1 mm. The shallower the crevice, the fewer are the chances for a possible development of pathological conditions within it. Judgment must also be exercised to guide us in determining what constitutes a normal and healthy gingival crevice, as, besides the factor of depth, the presence of inflammatory changes, the color and general appearance of the gingivae must be given consideration as well. A gingival crevice may become a pocket, lesion or pathological crevice as a result of inflammatory changes in the gingivae or in combination with detachment of fibres of the cenmental gingivae from the cementum. The subsequent developments in pocket formation are further detachment in the pericementum, with progressive decalcification, and loss of alveolar bone. The retrogressive changes within the periodontium, regardless of their inciting causes, are always attended by infective processes. A periodontal pocket permitted to run an unchecked course will, from the nature of its own forces, continue to progress apically until all normal structures within its path have become involved. Once a pocket is established, the beginning of a vicious circle has been started which becomes an aggravating factor in its further development. The mechanical characteristics of a pocket prevent the possibility of non-reinfection, for the reason that it cannot be kept free from irritating elements.

The elimination of the pocket should aim at the re-establishment of the gingival crevice to a normal depth, a zero one if possible. Such a depth provides the maximum safeguard against the possibilities of reinfection, as a shallow crevice minimizes the hazards. It should also aim at restoring to a state of health all parts of the periodontium in which pathological changes have left infective processes in their wake. The first desideratum may generally be accomplished in gingivitis or in incipient cases, where the decalcification of the alveolar crest has just started, by removing the irritating factor of either endogenous or exogenous origin. This should result in a reduction in the height of the gingivae through removal of inflammatory changes within the tissues. The second desideratum cannot always be brought about without a thorough removal of all diseased tissues to the level of healthy structures. The alternatives of this measure are the recurrence of the original condition and the leaving *in situ* of residual areas of infection.

In the symptomatic treatment of periodontal disease, different modes of therapy are advocated and employed. There can be no question but that each accepted method is valuable and has its place, either when used alone or as an adjunct measure. A clouding issue arises when any particular therapeutic means is recommended as being the only acceptable type for treating all phases and divisions of periodontal disturbances. This condition has undoubtedly been one of the major deterrents in the progress of periodontia by causing confusion in the minds of a large segment of the dental profession and a resulting apathy in this important branch of dental practice. Pro and con claims of so-called conservative and radical approaches to the problem are not helping to shed much light and encouragement to those whose only background in periodontia is the modest information they received on the subject while undergraduates.

The burden of responsibility for this lack of understanding and uniformity in methods of treatment is upon those who are looked upon as authorities in this field of dental endeavor. Instead of trying to straighten out their different viewpoints, many of them cling tenaciously to their individual tenets and refrain from examining ideas emanating from other sources. Until such time as this is done, or else until a new generation springs up endowed with more liberal ideas, periodontia is apt to remain in the same static position that it finds itself in today.

METHODS OF TREATMENT

The following means of treatment are employed for the purpose of altering the depth, contents and physical environment of the periodontal pocket or lesion.

What may be termed the simplest form of periodontal treatment is proper and adequate tooth-brushing, which is often sufficient by itself to correct certain types of gingivitis frequently due to deficient mouth hygiene. The proper use of a suitable toothbrush may be considered the alpha and omega of periodontal management, as upon it depends not only the correction of many common forms of gingival inflammation, but also the successful post-operative outcome of any mode of periodontal treatment. If the patient is not instructed as to the proper type of toothbrush and its correct application to meet the particular requirements of the case, failure may then be expected, regardless of the mode of therapy that was employed.

Various drugs are valuable in sub-gingival medication for the purpose of reducing the inflammatory changes within the tissues and the bacterial flora in the crevice or pocket. Aside from treatment in Vincent's infection, the use of drugs to inhibit suppuration alone is not indicated. Suppuration is generally found in pockets where a certain amount of detachment and loss of bone have taken place, and its stopping from medicaments can be but temporary. Drugs, such as aniline dyes, are invaluable in the treatment of various types of gingivitis, sometimes when used alone or else in combination with scaling. Vastine[3] and Krogh[4] have brought out the advantages of reducing the inflammatory and suppurative changes in periodontal lesions prior to other forms of treat-

ment. The principle of this measure is to reduce the bacterial flora in the pocket and diminish the inflammation within the soft tissues. This pre-operative procedure permits sub-gingival curettage, surgery or any other technique to be carried out in a cleaner field, and is conducive to better post-operative results.

Scaling or sub-gingival curettage performs the complete removal of all irritating elements within the crevice or pocket. It consists in scaling or curetting the calcareous deposits, the embedded ends of detached connective tissue fibres, the rough necrotic cementum and leaving a clean, smooth root surface. This procedure may be considered basic in most cases of gingivitis and in the treatment of shallow pockets. The internal lining of the outer wall of the pocket will become re-epithelized following re-organization of the underlying connective tissue.

In deeper pockets, where the extension of infective changes reaches into contiguous structures, the danger of leaving areas of chronic inflammatory tissue is ever present. Since a re-attachment to close the pocket does not occur, the mere apposition of epithelium against the denuded cemental surface does not prohibit calcareous deposits and other irritating factors from entering the treated pocket and starting new inflammatory reactions leading to re-infection.

The argument is often advanced that it is preferable to repeat sub-gingival curettage as often as may be deemed necessary, rather than resort to expedients that remove tissue in order to eradicate the lesion. Sub-gingival curettage in deeper pockets is a long and tedious procedure, and its frequent repetition cannot be considered with much favor by most patients. Furthermore, it is not always possible to detect the recurrence of infection, as suppuration and inflammation are not always observable. One of the principal reasons for the ineffectiveness of this type of treatment in other than mild forms of periodontal disturbances is its inefficacy to remove all infected processes contiguous to the pocket. Instrumentation, scaling or sub-gingival curettage when used alone is effective only in those cases where the shrinkage of the soft tissues of the crevice or pocket, following the subsiding of the inflammatory changes, will be sufficient to restore a sub-gingival sulcus of normal depth.

The flap operation brings about the same results as sub-gingival curettage. It offers a clear view of the affected areas, thus permitting a more rapid and thorough scaling and curettage, but does not eliminate the pocket beyond the point of its reduction produced through shrinkage or partial resection of the soft tissues.

GINGIVECTOMY

The principle of gingivectomy is to remove the soft tissues of the pocket to its bottom, where detachment ends. This technique succeeds in eliminating the soft tissues comprising the outer wall of the pocket, but does not remove the chronic inflammatory tissue that often exists between the bottom of the lesion and the alveolar bone, where it may be found embedded in its cancellations. This tissue is of infective nature, and when left in position becomes a residual area and has the tendency to proliferate. It is for this reason that gingivectomy procedures advocate the frequent removal of surgical dressings and the cauterization of the exuberant granulation tissue with silver nitrate or some other cauterant. Gingivectomy would seem indicated in mild types of periodontal disease, where the pockets are incipient and the infective processes are not apt to extend beyond the limits of the depth of the lesions.

When stages of periodontal involvement have advanced beyond the point where medication, sub-gingival curettage or gingivectomy cannot attain the desired objectives, it then becomes expedient to employ techniques that go beyond the limits of the pocket and eradicate all soft tissue to the level of the alveolar border.

Electro - cautery, electro - coagulation, chemo-therapy and surgery techniques provide the means of obliterating the periodontal pocket and all those infective processes that make up the disease entity termed

periodontoclasia. To say which mode presents the better approach is difficult, as the question resolves itself into a matter of personal choice. The essayist belongs to the surgical school of thought, as he believes that it furnishes the greater degree of accuracy and control.

From a mechanical standpoint, the elimination of the periodontal pocket is essential in order to reduce the chances of reinfection to a minimum. The total removal of the soft tissue, the "walls" of the lesions, allows the regeneration of a new gingival crevice often approximating zero in depth. Aside from the thorough removal of all infected tissue, the resurgence of a new, healthy gingival crevice of shallow depth is probably the most valuable feature of these techniques, whose principle embodies the total eradication of the periodontal pocket. Once established, this ideal crevice can generally be maintained in a state of health through adequate prophylactic measures.

There appears to exist in some quarters a certain amount of hesitancy and objections to tissue removal techniques. To some, such procedures would seem to imply disfiguration. To others, the drawback would be that exposed dentine is necessarily followed by carious disintegration of that tissue. The amount of uncovering of root surface following pocket obliteration is in proportion to the degree of progress of the disease. In cases where the alveolar destruction is not too far advanced, the results of tissue removal are scarcely noticeable. In more advanced cases the esthetics are often favored by the fact that the majority of oral cavities have a normal lip length which does not reveal the areas above the crowns of the teeth. Practical experience would show that from the patient's standpoint the acceptance of root exposure comes before having to submit to extractions or to leaving teeth in the mouth with potential infections. Clinical experience, on the other hand, would demonstrate that but a very small percentage of the cases following tissue removal subsequently have softening of the dentine, and that, furthermore, this same condition would have occurred without the institution of any form of treatment.

Thus far the attempt to justify the elimination of the periodontal pocket has been considered only from the physical side. In the appraisal of periodontal disease we are faced with two problems: first, the arresting of disintegrating changes within the supporting structures of the teeth, with the objective of prolonging the life and function of the dental organs; second, the eradicating of infective forces within the diseased periodontium, which constitute the most common and important oral menace to general health. The loss of teeth is always a serious matter, but secondary, however, in importance to that factor which is capable of affecting the entire organism. In the not too distant past, periodontal disease was generally overlooked as either a potential or an active force of infection, or at least was subordinated in importance to those infections related to periapical involvements. The realization is gradually materializing that periodontal disease overshadows all other forms of oral infection in degree of prevalence, potentiality and severity. Burket[5] is of the opinion that it prevails over more extensive areas and contains a heavier bacterial flora, which is composed of more potential pathological organisms. The intricate gingivae and dental periosteum are more vascular and have an extensive lymphatic system which favors the dissemination of bacteria or their products to other areas of the body. The same authority[5] also states that it has been estimated that even in mild forms of periodontal disease the total ulcerative area may exceed eight square inches. The same amount of similar diseased tissue in other parts of the body would readily be the cause for serious concern.

When a break occurs in the epithelial lining of the crevicular surface, an inflammatory change takes place in the underlying connective tissue. This is the initial reaction to bacterial invasion and part of the defense mechanism within the connective tissue. Box[6] has demonstrated that the successive stages in the evolution of a periodontal

pocket are followed by further bacterial invasion. He believes that tissue solution, resulting in the stripping of gingival tissue from the cementum, is due to the action of ferments liberated by the poly-morphonuclear leucocytes or by bacteria. Bacterial infection may therefore be considered a major factor in every stage of the course of periodontal disease.

The internal surface of the soft tissue component of the pocket establishes periodontal lesions as ideal absorptive channels of infection. This tissue, when partially or completely stripped of epithelium, exposes the underlying connective tissue with its rich vascular circulation, through which bacteria and their toxins have ready access to other portions of the body.

The most offending bacteria in periodontal infections is the *Streptococcus viridans*, which Cook and Stafne[7] and others have isolated from deep pockets and grown in glucose brain broth injected intravenously into rabbits, and have demonstrated its specific power of elective localization.

Besides the direct access of bacterial invasion within the periodontal lesion, there is also the intimate relation of bacterial exudates from the pockets with the gastro-intestinal and respiratory tracts.

The systemic disorders that may arise or become aggravated from periodontal lesions are similar to those pathological conditions that may develop from any other source of focal infection. Aside from the possibility of forming secondary manifestations, periodontal lesions may produce a lowering of the vital forces or resistance through overloading the system with toxic substances. Following the eradication of periodontal infection there generally will be found an increase in vitality manifesting itself in a feeling of greater energy and general well-being. Clinical experience would demonstrate that such results, as well as the cleaning up of symptoms of systemic disorders, are of common occurrence in periodontia practice.

There does not exist at the present time any positive index to determine whether a focus of infection, wherever it may be located, is producing secondary manifestations elsewhere in the body. In the absence of such an indicator, and in the light of our knowledge of the metastatic possibilities of focal infection, periodontal disease in all of its stages must be regarded as a serious systemic hazard.

SUMMARY

The rationale of the elimination of the periodontal pocket in the treatment of periodontal disease may be summarized by the following basic points:

First: the physical characteristics of the periodontal pocket render it the ideal medium for bacterial propagation leading to infective changes within its confines. The disappearance of the pocket, resulting in a shallow crevice, affords the maximum possibility against re-infection.

Second: Periodontal disease, as the primary potential or active focus of infection in the oral cavity, must be considered at all times a serious menace to general health. It follows that all infective processes attending its degenerative course must be removed, with a return of all tissues to a state of health.

These objectives may only be achieved through the complete elimination of the pocket.

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Some Recent Concepts of the Connective Tissue Ground Substance and Their Implications in Dental Disease

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THE EXTRACELLULAR interfibrillar matrix of the connective tissues is termed the ground substance. This optically homogeneous material is largely a carbohydrate-protein complex (glycoprotein) which is thought to be more or less highly polymerized[1] as in the hard substance of bone and dentin or in the loose gel of embryonal connective tissue. The ground substance is altered at the interphase between cellular layers and connective tissue as in skin, capillaries and glandular organs to form with the embedded fibers, the "basement membrane." Gersh and Catchpole[1] have shown that the ground substance is very labile, responding in a most sensitive manner, to physiological and pathological states.

The dental tissues, with the exception of the enamel, originate from the connective tissue. The matrices of dentin, cementum, bone and of the periodontal soft tissues are also largely glycoprotein polymers.[2,3] In diseases of these tissues the ground substance is profoundly altered and it is believed that these changes are mediated by enzymatic mechanisms. Some of the changes occurring in dentin caries and in a periodontal disease, desquamative gingivitis, will be described here.

In studying these tissues some newer methods have been used. Preparations which were to be studied by histochemical methods were fixed by the Altman-Gersh freezing-drying method. Carbohydrate-protein complexes were visualized by the periodic acid-Leucofuchsin procedure.[4] In certain instances tissues were assayed for hyaluronidase and collagenase activity.

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In caries of the dentin the reactions of sections of the dentin with the periodic acid-leucofuchsin reagent is intense.[5] This is interpreted as evidence of depolymerization of the ground substance. Since dentin which has been previously treated with acid can be softened and dissolved by an enzyme "collagenase" it was suggested that the changes occurring in dentin caries might be on a similar basis. This view was confirmed by assay of extracts of carious dentin. The presence of collagenase and hyaluronidase like activities were demonstrated. These findings have led to the conclusion that in caries of the dentin, the dentin ground substance is depolymerized in part by the action of a "collagenase." The hyaluronidase is believed to play a role in the pulpal reaction frequently associated with deep caries.

Striking changes in the ground substance occur in desquamative gingivitis.[4] We were able to investigate eight patients; six of the group were women. Biopsies were obtained and studied employing histochemical procedures. The connective tissue ground substance was profoundly altered; large portions of it were dissolved. The basement membrane was either absent or attenuated. When compared to normal tissues the ground substance was shown to contain increased quantities of water soluble glycoprotein and alcohol insoluble water soluble glycoprotein residues. In some areas the cementing substance between the epithelial cells was dissolved.

It is suggested that these changes are due to the production of abnormal quantities of enzymes by the connective tissue cells which then depolymerize the ground substance and cementing substance. The predominance of female patients in our group and in those series reported by other investigators suggest an influence of the female sex hormones on the enzymic activity of the connective tissue

cells. This is comparable to hormonal effects in the connective tissue reported by Gersh and Catchpole.[1]

The concepts cited here may be extended with a little imagination to the explanation of other pathologic and physiologic processes of dental interest.

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Occurrence of Gingivitis in Suburban Chicago School Children

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GINGIVITIS is a widely distributed oral affliction. Many studies have reported on its etiology and pathogenesis. However, only a few have attempted to measure the extent and incidence of the disease in various age groups. The reason has been the lack of a uniform system of classification and examination for the assessment of gingivitis in large population groups such as the one developed for the assessment of caries in large groups (DMF Teeth; DMF Surfaces) (Klein and Palmer, 1941).

The need for a simple, quantitative method of indexing and assessing gingival disease has been emphasized by King (1945) and by Schour and Massler (1947). Such a method should satisfy the following criteria: (1) simplicity, (2) speed, (3) accuracy, (4) quantitativeness, and (5) reproducibility. The method suggested by Schour and Massler (1948), which they termed the P-M-A Index, satisfies these criteria.

While a number of studies have attempted to determine the extent of gingival disease in adults, very few have been concerned with this problem in children. The tendency has been to regard gingival disease as a disease of adults rather than of children. However, the groundwork for adult disease is often laid in childhood. It was deemed important to ascertain the frequency of gingivitis in normal, healthy children, as well as its extent and severity. A baseline might thus be established for comparison with groups of older ages or of different economic, geographic or nutritional status. This study was, therefore, undertaken to determine the occurrence of gingival disease in grade-school children, using the P-M-A method of indexing.

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REVIEW OF LITERATURE

Table 1 summarizes the data reported by various investigators on the prevalence of gingivitis in children. It is very difficult to compare these findings, since different methods were employed to obtain them and the criteria of assessment were often varied.

Ainsworth and Young. Ainsworth and Young (1925) made the first serious attempt to determine the extent of gingival disease in children on a public health scale. They assessed the occurrence of gingivitis in 4,063 children from 2 to 14 years of age, residing in England and Wales. Ainsworth and Young used a simple method of assessment, classifying each child as having (1) no gingivitis, (2) slight gingivitis, (3) intermediate gingivitis and (4) severe gingivitis. The slightest deviation from the ideal firmness and paleness of perfect health was recorded as "slight gingivitis." The term "severe gingivitis" was restricted to those cases showing severe hyperemia and swelling of the gingival margins with some tendency to bleed. The intermediate grade included all cases of gingivitis which could not be definitely classed under either of the above headings.

They observed no gingivitis in 2,438 children (60%), slight gingivitis in 1,439 (35%), intermediate gingivitis in 244 (6%) and severe gingivitis in 36 (0.8%). Their study showed only a negligible difference between boys and girls although there was a suggestion that boys had a slightly higher incidence than girls in the later age groups.A definite increase in the number of children with gingivitis was evident with increase in age, from 18.5% at 5 years to 53% at 14 years. Such increase was found not only in the number of children affected but also in the severity of the gingivitis.

Messner et al. In the United States, Messner et al. (1936) reported the results of a questionnaire type of survey of the occurrence of gingivitis in children. They collected the figures (usually estimated) which were submitted by dentists from 26 states and published the data on 1,438,318 children (boys and girls) from 6 to 14 years of age. *They made no attempt to differentiate among the various forms or degrees of gingivitis.* Their data indicated that in white children (1) the incidence of gingivitis increased with age from 3.5% affected at 6-8 years, 5.6% at 9-11 years, to 8.6% affected at 12-14 years, and (2) the occurrence of gingivitis was higher in boys than in girls (6.4% and 4.8% respectively).

Day. In India, Day (1940) noted the occurrence of gingivitis in 756 children of one school in Lahore. The average age was 13.58 years. He distinguished only between slight and severe gingivitis. He observed that 38.09% showed slight gingivitis and 30.69% showed severe gingivitis. Approximately 31% are presumed to have exhibited no gingivitis.

Day (1940) also examined 203 children of one Kasur school in India, which was in a fluoride endemic area. He found no gingivitis in 82 (40.4%), mild gingivitis in 74 (36.4%), moderate gingivitis in 44 (21.7%) and severe gingivitis in 3 (1.5%). The incidence of gingivitis increased with age from 30.0% at 6-7 years to 69.51% at 9-12 years and 69.01% at 13-18 years. He concluded that the occurrence and severity of gingivitis was greater in the Kasur fluoride-endemic area than in the non-endemic Lahore area, a conclusion which, while supported by other observations, is not supported by his figures quoted above.

Day (1944) also surveyed the prevalence of gingivitis in five villages near Hissar City. These villages were affected the worst in the famine district. The average age of the 314 children examined was slightly under 12 years. A remarkably high incidence of gingivitis was observed in all villages. Eighty-five percent of the children showed mild to severe gingivitis of the hypertrophic and non-hypertrophic types. Seventy-eight percent of the children suffered from hypertrophic gingivitis. A generalized hypertrophic gingivitis is rare in children of the United States. In these malnourished children of India it occurred usually as a hard, fibrous and painless swelling characterized by more or less uniform thickening or hyperplasia of the marginal gingivae. From Day's observations it appeared that the occurrence of gingivitis increased greatly with undernutrition and malnutrition.

Day and Shourie (1944) examined 613 healthy Indian school children 5 to 15 years of age. In the group 5-10 years of age, 74.4% showed gingivitis and 19.8% were free of gingivitis. In the group 11-15 years of age, 73.2% showed gingivitis and 20.8% were normal.

Bruckner. Bruckner (1943) examined 1634 American school children 4-16 years of age. Gingivitis was assessed as either "slight" or "severe." Pupils revealing a mild marginal inflammation with a slight tendency towards bleeding and with a history of occasional hemorrhage on brushing were recorded as "simple" or "slight" gingivitis. Those showing a deeper seated inflammation with puffiness of the gingival margins and with interproximal involvement were classified as "severe." Of the 819 boys examined, 735 (89.7%) showed healthy gingivae, 72 (8.8%) slight gingivitis and 12 (1.5%) severe gingivitis. Of the 815 girls examined, 757 (92.9%) showed healthy gingivae, 47 (5.8%) slight gingivitis and 11 (1.4%) severe gingivitis.

King and Francklyn. King and Francklyn (1944) observed two types of gingival lesions in the Gibraltar children evacuees in London. Any deviation of the gum from the ideal, pale pink color and firm, close adherence to the teeth was recorded as gingivitis. They differentiated between marginal gingivitis (M type lesions) and papillary gingivitis (P type lesions). In cases with marginal inflammation, the interdental

TABLE 1
Occurrence of Gingivitis in Children According to Different Investigators

Group Studied	*Number Examined*	*Age Group (in yrs.)*	*Percent of Persons*					*Author*
			Free	*Affected*	*Mild Gingivitis*	*Moderate Gingivitis*	*Severe Gingivitis*	
School Children in England and Wales	4,063	2-14	60%	40%	33.2%	6.0%	0.8%	Ainsworth and Young (1925)
Children in 26 States of U. S.	1,438,318	6-8 9-11 12-14	96.5% 94.4% 91.4%	3.5% 5.6% 8.6%				Messner, et al (1936)
Middle Class Children in India	756	13.8	31%	69%	38.0%		30.7%	Day (1940)
Fluoride Endemic Area in India	203	5-18	40.4%	59.6%	36.4%	21.7%	1.5%	Day (1940)
Famine District in India	314	Under 13	15.3%	84.7%				Day (1944)
Various Socio-Economic Levels; 14 Nationalities (U. S.)	819b 815g	4-16 4-16	89.7% 92.9%	10.0% 7.1%	8.8% 5.8%		1.5% 1.4%	Bruckner (1943)
MiddleClass India) (IslamiaHighSchool)	1054b	9-17	0.6%	99.4%	25.8%		73.6%	Day and Shourie (1947)
English Children Gibraltar Evacuees	137b 266b 135	11-14 11-14 10-14	22.6% 12.4% 14.8%	77.4% 87.6% 85.2%				King and Francklyn (1944)
Dundee Primary School Children (English)	43b 60g	12-14 12-14	11.6% 10.0%	88.4% 90.0%				
Harpenden Institution (English)	40b 40b 51b 39	12-14 12-14 11-14 11-14	7.5% 2.5% 20.4% 43.6%	92.5% 97.5% 79.6% 56.4%				King (1945)
4 Communities in Italy, Suffering from Malnutrition	682 721	6-10 11-20	59.7% 44.7%	40.3% 55.3%	32.4% 48.3%	6.6% 6.7%	1.1% 0.3%	Schour and Massler (1947)
2 Chicago Suburbs	804	5-14	35.7%	64.3%	35.9%	12.3%	9.2%	Present Study

*b—boys g—girls

papillae were not unduly involved. In the papillary gingivitis a glazed reddish swelling of the interdental papillae was observed.

The incidence of gingivitis in the Gibraltar children was compared with the incidence in English boys of almost the same age and similar basal diet. Gingivitis, in general, occurred with similar frequency in English and Gibraltar children (77-79% and 85% respectively). However, when types were differentiated, a striking contrast between the English and the Gibraltarians was noted: only 1-2% of the former suffered from papillary lesions, compared with 40% of the Gibraltar children. Of the English children, 75-86% suffered from M type lesions, as compared with 45% of the Gibraltar children.

Up to this time (1944), no serious attempt to determine the amount of gingivitis quantitatively in any given mouth had been made. Such a device would be essential before any serious attempt could be made to evaluate the incidence of the disease quantitatively. The crude device of distinguishing between no gingivitis and mild, moderate or severe gingivitis does not lend itself to quantitation. A concrete figure of objective measure cannot be assigned to such subjective evaluations as mild, moderate or severe. The majority of studies merely attempted to determine the percentage of the persons affected and those free from gingival disease. In addition, most studies used arbitrary and subjective methods of evaluating mild, moderate and severe gingivitis. While these studies served to pilot the investigation of the epidemiology of gingivitis, comparisons obtained by different investigators are not valid, since each used different, non-quantitative criteria of evaluation.

King. King's study in 1945 showed for the first time that it might be possible to define mild, moderate or severe gingivitis in objective and quantitative terms and figures. He limited his observations to the gingiva in the incisor and canine regions and divided these regions into eight units (see Fig. 1). The tissue between the central incisors of each jaw was excluded because of interference by the frenum. Each gingival unit was examined and recorded separately.

He considered the gingiva as normal (0) when the gum was pale pink in color and the margin tapered onto the tooth surface and filled the interdental space. Slight disease (1+) was evidenced by swelling and glazing of the gum margin with or without injection. This change was localized almost entirely to the interdental papilla. The papilla became slightly blunted or rounded. Moderate disease (2+) was evidenced by swelling and pocket formation on the buccal or lingual aspects. The tissue often became spongy in texture and exhibited spontaneous hemorrhage or even more profuse bleeding on pressure. Severe disease (3+) was considered if the above change increased in degree.

King conducted a survey of primary and secondary school children, 12-14 years of age, young factory

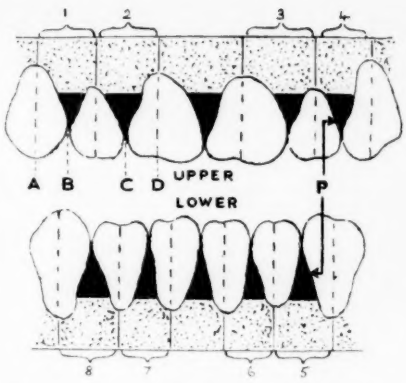


Fig. 1. Diagram illustrating use of gingival unit as employed by King (1945). (Fig. 1 from King, 1945.)

workers and expectant and nursing mothers, living in Dundee. He also examined primary and secondary school-age children at a Harpenden Institution, using this method of assessing the gingiva.

In the 90 children 11-14 years of age at the Harpenden Institution, gingivitis was present in 70.0%, and absent in 30.0%.

Both the incidence and extent of disease were significantly greater in the children from lower economic levels than in those of higher economic levels. He observed, too, that boys in either category had more gingivitis than did the girls.

The significance of this study lies not so much in the figures presented as in the fact that a quantitative method of evaluating the incidence and extent of gingival disease was presented for the first time.

Day and Shourie. Day and Shourie (1947) examined boys and girls of the Islamia High School and Queen Mary College, Lahore, India. At the Islamia High School, which is a middle-class boys' school, 1,054 subjects from 9 to 17 years were examined; in Queen Mary College, which is attended mostly by girls from 5 to 24 years and some boys 5 to 11 years (the sons and daughters of ruling princes and well-to-do families), 323 subjects were examined, the group being made up of 261 girls and 62 boys.

The clinical signs used for assessing the presence and the severity of gingival disease were (a) *surface*, disappearance of matte surface with reduction or absence of stippling; (b) *gum margin*, swelling, color change, detachment from tooth surface with deepening of the gingival crevice; pocket formation and, in late stages, suppuration; sometimes reddening and ulceration; (c) *papilla*, swelling and color changes, which may be confined to papillae or associated with similar changes in other parts of the gum margin;

blunting of the normal pointed papillary tip detachment from the adjacent teeth with interdental pocketing. Hemorrhages may occur from any part of the gingival margin, including the papillae, either spontaneously or on pressure.

The degree of involvement and severity were classified as (1) slight to moderate disease, (2) severe disease and (3) very severe disease; denoted by 1+, 2+, 3+ gingivitis, respectively. In order to obtain a satisfactory basis for the comparison of the gingival status in India and elsewhere, the criteria suggested by King (1945) for gingival health and disease were followed as closely as possible.

Day and Shourie compared their findings with the statistics available in the literature and concluded that the incidence of gingival disease in India, at least in the lower and middle classes, was extremely high and probably the highest so far reported in a similar age group. They noted that only 6 of the 1,054 children (0.57%) examined of Islamia High School (middle-class persons) showed an absence of gingival disease. In all groups, the incidence of gingivitis was practically 100% (94.7-100%). They also showed that 73.62% of the cases showed either severe or very severe gingival disease, 25.81% showed slight to moderate gingival disease and only 0.57% of the children could be classified as having normal gingiva.

In Queen Mary College where 261 girls from the wealthy class were examined, both the incidence and severity of the disease were significantly less than in the Islamia High School. When the same age groups (9 to 17 years) from both places were compared, it was observed that the incidence of disease was 99.43% for the 1,054 boys and 73.74% for the 179 girls. On the other hand, if the 58 boys of the some social and economic status were compared with comparable 106 girls from the same class, they found that in the same age range (6 to 11 years) both the incidence and the extent of the disease were greater in the boys than in the girls, the incidence of gingival disease being 77.59% in boys and 70.75% in girls.

Schour and Massler. In 1947, Schour and Massler conducted a survey of the occurrence of gingivitis in post-war Italy in various age groups. The method of assessment of gingivitis was as follows:

0="no inflammation in any region of the mouth."

1+="mild gingivitis; localization of inflammation usually to the interdental papillae or free gingival collar of 1 to 3 of 6 lower anteriors."

2+="moderate gingivitis, extension of inflammation to form a more or less continuous band along the marginal gingivae of more than three regions of teeth."

3+="severe gingivitis, extension of inflammation from the marginal gingivae into the attached gingivae, affecting almost all anteriors and some posteriors."

4+="very severe gingivitis, where Vincent's infection and other more severe stomatitis were included."

The prevalence of gingivitis in four communities in Italy was determined from the examination of 3,905 persons aged 6-60 years. These persons suffered from moderate to severe degrees of malnutrition. It was observed that (1) the incidence increased with age and ranged from 40% in the group 6-10 years old to 98% in the group 51-60 years old; (2) the severity of the gingivitis also increased with age, starting with a mild papillary type in the group 6-10 years old, extending through the marginal gingivae in the middle-age group and terminating in the attached gingivae in the 51-60 years old group.

As a result of this study, Schour and Massler in 1948 suggested a quantitative method of assessing gingival disease which they termed the P-M-A-Index. The method is used in this study.

SUMMATION OF THE LITERATURE

A review of the literature cited above indicates that a number and variety of attempts have been made to assess gingivitis, using different methods. It also shows that the methods of assessment became refined, more objective and more quantitative as the number of studies increased.

In the beginning, clinicians (practitioners) attempted only to distinguish between the different types of gingival disease and to present only *qualitative* diagnostic criteria which would enable them to classify (and eventually to treat) the various forms of gingival disease.

The second era was ushered in when the clinician became interested in the public health aspects of gingival disease and made serious attempts to determine the *extent* of gingival disease among the various population groups.

Ainsworth and Young (1925) first attempted to differentiate between children with no gingivitis and children with gingivitis. They also classified gingivitis as slight, medium and severe, but made no serious attempts to employ a quantitative method. Their classification was entirely subjective, but this fault was not corrected until 1945 when King presented his method of counting the 8 anterior gingival units. It is surpris-

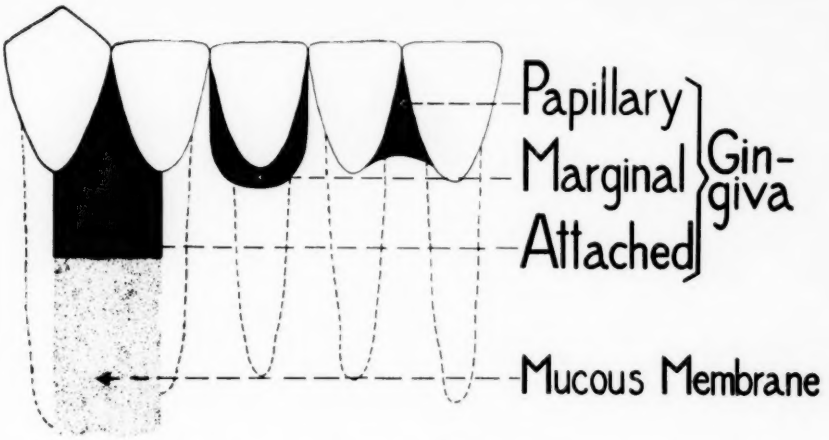


Fig. 2A. Diagram illustrating use of gingival unit (P-M-A) as employed by Schour and Massler (1947). (Fig. 1 from Schour and Massler, 1947.)

ing that between 1925 and 1945 no marked improvement of Ainsworth and Young's method was presented.

It was only very recently that King (1945), and Schour and Massler (1947) attempted to define mild, moderate and severe types of gingivitis in a more precise and objective manner. The gingiva was for the first time divided into gingival units, and the papillary gingiva, marginal gingiva and attached gingiva were clearly differentiated.

METHOD

The P-M-A Index. Previous studies have served only to give a general indication of the gingival status of the various groups examined. A more precise assessment is obtained if the recording is made of both the incidence and the extent of the disease by counting the number of gingival units (papillary or marginal) affected by disease.

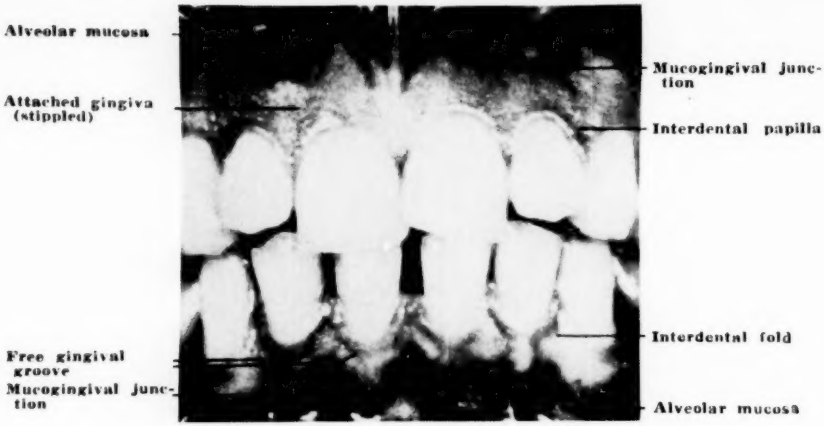
King (1945) proposed a semi-quantitative method of evaluation which was refined towards more exact and objective quantitation by Schour and Massler (1949). According to this method the gingiva is divided into gingival units consisting of the following parts (see Fig. 2A):

(1) The interdental papilla (P)—that portion of the gingiva that occupies the interdental space between two adjacent teeth.

(2) The marginal gingiva (M)—that portion of the gingiva which forms a collar of free gingiva around the neck of the tooth. Being free, this portion of the gingiva lies coronal to the bottom of the gingival sulcus.

(3) The attached gingiva (A)—that part of the gingiva which is attached by a dense fibrous matte to the underlying alveolar bone. It is separated clinically from the marginal (free) gingiva by a distinct line, the free gingival groove, and from the mucous membrane by the muco-gingival junction (Orban, 1948).

Schour and Massler (1948) indicated that inflammatory lesions tend, for the most part, to begin in the papillary gingiva and to spread from that area into the marginal gingiva. In severe gingivitis the inflammation



Surface of the gingiva of a young adult.

Fig. 2B. Figure illustrating the contour of gingiva in a case of no gingivitis (Fig. 1, from Orban, 1948).

then spreads to the attached gingiva (periodontitis). The degree or severity of the inflammation, therefore, is usually indicated also by its location, mild gingivitis being confined to the papillae, moderate gingivitis involving the marginal gingiva and severe gingivitis affecting the attached gingiva.

It, therefore, was logical to examine the (1) papillae, (2) labial margins and (3) attached gingivae and to record (1) papillary gingivitis, (2) marginal gingivitis and (3) attached gingivitis in that order.

Schour and Massler also indicated that inflammatory changes are characteristic of the young and that non-inflammatory, atrophic changes are characteristic of older age groups. Thus, this study is confined to the problem of gingivitis, other periodontal disease (gingivosis, periodontosis, recession) being practically non-existent in our groups.

Criteria of Assessment. Since the various types of gingivitis are not clearly defined, it was decided to adopt a classification of severity which would avoid ambiguity and would be based on naked-eye appearance. The severity of inflammation was, therefore, graded numerically in successive degrees according to increasing intensity and extent.

0 = No gingivitis, with the following characteristics:

Color: pale pink.

Contour: see figure 2B.

Texture: firm, no bleeding on firm digital pressure.

Tonus: close adherence of the free gingiva to the teeth just coronal to the cemento-enamel junction and of the attached gingiva to the alveolar bone. When two adjacent teeth have their interproximal surfaces in direct apposition, the gingiva should completely fill the interdental space; the dental margin of the gum should taper off to the enamel.

1 + = Mild inflammation, with slight change in color and little loss of contour.

2 + = Moderate inflammation, with swelling, glazing and redness. Tendency to bleed on slight pressure. Papillae or margins become blunted or rounded in contrast to the normal tissue. Slight extension of the inflammation to adjacent tissue (P-M-A).

3 + = Severe inflammation, with more swelling and redness, pocket formation, spontaneous bleeding. Involvement of the adjacent tissue definite. Slight degeneration.

4+=Very severe, any degree more severe than above, including ulceration and sloughing (as in Vincent's infection).

Method of Examination. Each child was seated comfortably in an ordinary chair to which had been attached a portable head rest. The mouth was examined with a tongue blade using good natural or artificial lighting.

Two persons previously trained in the method of examination and in the criteria of assessment formed one team, one acting as examiner and the other as recorder. The recorder was seated face to face with the examiner and the child, so that he could at all times follow the action of the examiner. At regular intervals (approximately every 10th child) the examiner and the recorder interchanged so as to reduce fatigue and to lessen subjective opinions or trends on the part of the examiner. Gingival tissue of only buccal and labial surfaces was recorded.

Method of Recording. Each examination started from the lower left arch and was carried out to the lower right side, and similarly in the upper arch. The gingival papilla between second and third molars was counted as 7 and between first and second molars as 6, and so on. All the papillae were recorded, including those between the central incisors.

METHOD OF ANALYSIS

All data were recorded on a sheet similar to that shown in Figure 2C. Each papilla, margin and attached gingival unit which was affected was recorded as 1+, 2+, 3+ or 4+. However, in computing the P-M-A index, these figures, which indicate the severity of the effect, were disregarded. The average P-M-A for the group was determined by totalling the number of gingival units affected and dividing by the number of cases examined. The severity of the effect in each unit was not included in the statistics. Finally, the distribution of the cases into various degrees of gingivitis was made according to the following scheme:

	P-M-A
Absence of Inflammation	0–0–0
Mild (average or less)	1–0–0 to 2–1–0
Moderate (average to twice average)	3–2–0 to 5–2–0
Severe (more than twice average)	6–2–1 to 8–3–1
Very Severe	9–3–1 and above

The average P-M-A figure (2–1–0) was chosen for the upper limits of the category designated as "mild" since this would serve to include all the "normal" or "usual" cases. The category termed "mild" might be questioned by some who might prefer to include such very mild inflammatory changes within the range of normality. Since no criteria of "normality" exists, it was assumed that those cases which were below the average in gingivitis experience could be described as "mild." Those cases with more than average to twice average values were described as "moderate." Anything above that value was termed "severe." Unusually high values (less than 10 percent of the total) were termed "very severe."

MATERIAL

This study was based on the examination of 804 children; 381 from the Melrose Park Lutheran School and 423 from Mooseheart. The children ranged in age from 5 to 14 years and included an approximately similar distribution of boys and girls (see Table 2). Age was considered in years as of the last birthday.

"The Mooseheart children, as a group, are undoubtedly above the average physically. Only physically normal children are admitted to the orphanage and an extensive program of 'preventive medicine,' including a selective diet, regular sleep, and regular recreation, (both in and out doors) contributes to an excellent record of physical standard and health. The death record has been only one-tenth of one per cent during the last 25 years." (Reymert, 1947.) Dental care was well above average and, in fact, almost ideal, since there were available two

NAME: NO.:

SEX: AGE: EXAMINER:

RESIDENCE: DATE:

GINGIVITIS: P-M-A

PERM.	7	6	5	4	3	2	1	1	2	3	4	5	6	7
DECID.			E	D	C	B	A	A	B	C	D	E		
A														
M														
P														
P														
M														
A														
DECID.			e	d	c	b	a	a	b	c	d	e		
PERM.	7	6	5	4	3	2	1	1	2	3	4	5	6	7

ORAL HYGIENE	GOOD	FAIR	POOR

DEGREE

PALLOR	
INFLAMMATION	
ATROPHY	
HYPERPLASIA	
NECROSIS	

DEVELOPMENTAL ANOMALIES

Hypoplasias —

Hypocalcification —

Peg Teeth —

Impacted Teeth —

Fig. 2C. Diagram showing the sheet used for recording the data.

full time dentists and one-part-time dentist plus a well-equipped two-chair dental office. A study of the diet of the Mooseheart child by Johnston in 1943 showed that the diet of every child was excellent and that there were more children (4 out of 21) in excellent nutritive condition than are found in the usual public school.

The Melrose Park School is a non-residential Lutheran School for the working middle-class families. There is no dental clinic in the school, but a dentist makes regular examinations and treatments. The school claims better than average dental care for its children.

AVERAGE INCIDENCE AND GINGIVITIS EXPERIENCE

Percentage of Children Affected. Table 3 and Figure 3A show that 64.3% of the 804 children examined (5 to 14 years of age) showed one or more of their gingival papillae affected by gingivitis. Thirty-five and seven-tenths per cent of the children were entirely free from gingivitis. Of the 64.3% showing gingivitis, 35.9% showed mild, 12.8% moderate, 9.2% severe and 6.5% very severe gingivitis (Table 3 and Figure 3B).

The percentage of children affected with gingivitis increased significantly with increasing age. It increased very rapidly from 9.1% at 5 years to 69.3% at 7 years. The percentage rose slowly thereafter to a peak of 80% children affected at 11 years.

Average P-M-A per Child. The average P-M-A for the 804 children also increased with age. It was practically zero (0.16–0–0) at 5 years, but increased sharply to 8 years of age (3.1–1.2–0). After 9 years, the rise in average P-M-A was much slower but was constant and steady (Fig. 3A). Peaks in the average P-M-A could be noted at 8 years, 11 years and 13 years of age, with the depressions at 9 years, 12 years and 14 years of age. It is not clear whether the peaks and depressions are artifacts caused by the small numbers examined at each age.

Severity of Gingivitis. Table 3 shows that not only the incidence of gingivitis increased with age, but that the severity of the disease similarly increased with age.

TABLE 2
Age and Sex Distribution of 804 Children Examined

Age in Years	*Number Boys*	*Number Girls*	*Total Number*
5	25	30	55
6	30	30	60
7	40	25	65
8	40	44	84
9	48	47	95
10	48	27	75
11	43	47	90
12	74	41	115
13	46	43	89
14	44	32	76
5 to 14	438	366	804

It was interesting to note the distribution of children according to the severity of their gingivitis. Approximately 36 percent showed no gingivitis at all, i.e., clinically ideal gingivae. An equal number (36%) showed a mild degree of gingivitis (one to two papillae affected), which, while not ideal, were well within the range of clinical "normality." Thirteen per cent of the children were classified as having a moderate amount of gingivitis (3–2–0 to 5–2–0), three to five papillae and two gingival margins being inflamed. Clinically this group cannot be considered as seriously diseased nor is it perfectly "normal." Fifteen per cent of the children exhibited a clinically undesirable degree of gingivitis (severe and very severe). Nine per cent showed six to eight inflamed papillae and two or three inflamed gingival margins. Six and one-half per cent of the children had more than nine papillae and three

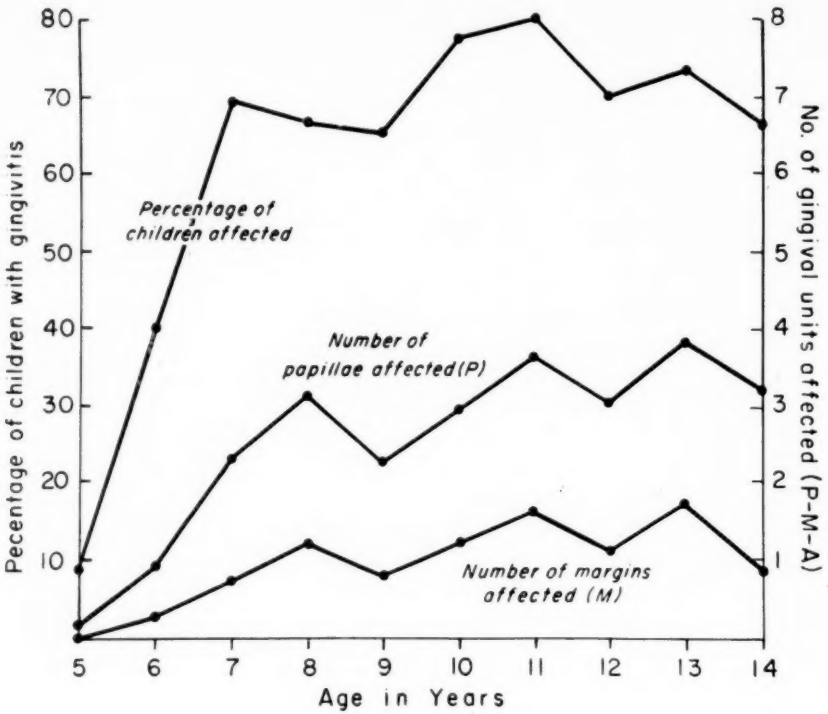


Fig. 3A. Graph showing increase with age in incidence of gingivitis (percentage of children affected) and in gingivitis experience (papillary and marginal) in 804 children 5-14 years of age.

margins affected. These figures are not inconsiderable, but the gingivitis is probably related to the process of eruption of the teeth rather than to any serious systemic factor.

Gingivitis and Eruption of Teeth. The character of the inflammation as observed clinically left us with the impression that the sharp rise in gingivitis observed at 7-8 years of age was caused primarily by the eruption of the four incisor teeth and the first permanent molars.

The sharp rise in percentage of children affected with gingivitis and in the number of papillae and margins affected with gingivitis at 7-8 years, as shown in Fig. 3, tends to support this hypothesis. The slower increase in gingivitis thereafter is probably related to the slower eruption of the bicuspid teeth and the fact that these latter teeth appear over a much longer period of time (9 to 12 years).

SEX DIFFERENCES

Percentage Affected with Gingivitis. Table 4 and Fig. 4 show that there was no striking difference between the percentage of the boys and the girls affected by gingivitis. Table 5 and Fig. 5 show that there was a slightly higher percentage of girls affected from 5 to 8 years of age, while boys showed a higher percentage affected from 9 to 14 years. The differences between boys

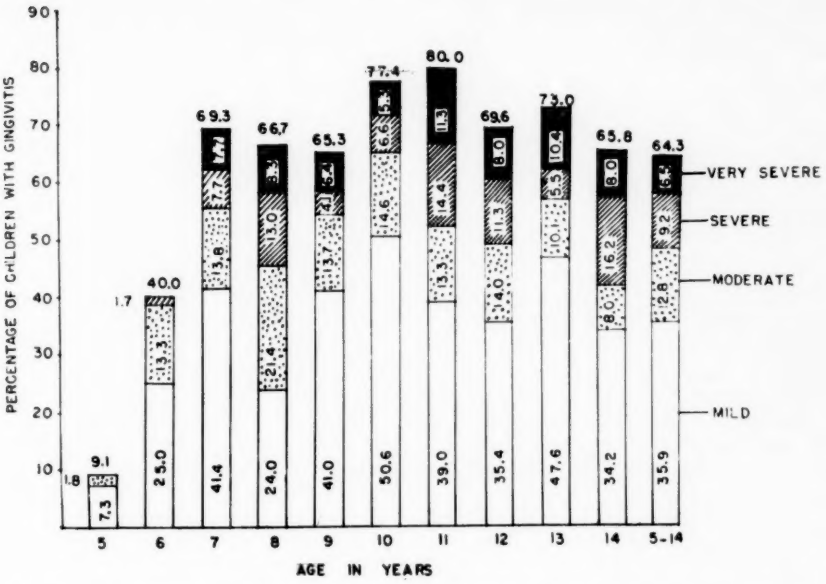


Fig. 3B. Histogram showing distribution of different degrees of gingivitis at different ages (804 children, 5-14 years of age).

and girls during these two age periods were tested statistically and found to be significant ($P = .02$).

Average P-M-A per Child. The average number of papillae affected (papillary gingivitis) for the total number of 438 boys was higher by 0.5 papillae than the average in the total of 366 girls (Table 4). The difference in marginal gingivitis (M) was even smaller (0.26 margins).

Boys also showed 0.26 more margins affected than did the girls (Table 4 and Fig. 4). There was also a greater percentage of boys (20%) with severe and very severe gingivitis than girls (13.1%) (Table 4).

However, if boys and girls are compared at *different ages*, it becomes apparent that the curve showing the number of papillae affected almost exactly parallels the curve indicating the percentage of children affected, i.e., girls have more papillary gingivitis at 5 to 8 years while boys have more papillary gingivitis from 11 to 14 years (Fig. 5).

When the number of margins affected are compared in boys and girls, it is clear that boys have consistently a greater number of margins affected (marginal gingivitis) (Fig. 5). The difference, although not great, is consistent.

These differences in the number of papillae and margins affected in boys and girls were tested and found to be significant ($P = .02$).

DISTRIBUTION OF GINGIVITIS IN THE ANTERIOR AND POSTERIOR SEGMENTS OF THE MOUTH

It became fairly obvious during the course of examination that a great proportion of the gingivitis observed in any given mouth was confined, for the most part, to the ten anterior (upper and lower) gingival units. These anterior segments experienced by far

the greatest attack by gingivitis. King in 1945 had suggested the examination of only the anterior gingival segments rather than the entire mouth for this reason. He did not, however, present any analysis or definite figures to justify this procedure. In view of this fact, an analysis of the distribution of gingivitis in the different segments of the mouth, especially in the anterior segments, was undertaken.

The use of the ten anterior units as an index to the gingivitis experience of the entire mouth would be desirable for a number of reasons. First, from an epidemiologic point of view, such procedure would save considerable time in examination with the result that larger numbers of persons could be examined. Second, it is much easier to observe the anterior segments than the posterior segments, so that greater accuracy and, therefore, uniformity in observations would be attained. Third, and probably the most important, if large groups are to be compared, it is more accurate statistically to compare comparable segments of the mouth rather than the entire mouth.

In order to compare the gingivitis experience of the anterior segments with that of the entire mouth, the data were reanalyzed as if only the ten anterior gingival units had been examined. The results thus obtained were then compared with the findings of the entire mouth, already presented.

Percentage of Children with Gingivitis. If the percentage of children with gingivitis is calculated from the examination of the anterior segments alone, the figures thus obtained for different age levels do not differ very markedly from the figures obtained from the examination of the entire mouth (Table 6A, compare Columns 3 and 5). In fact, a remarkable parallelism between the two sets of figures thus obtained exists at practically all age levels (Fig. 6).

The difference in the figures obtained by the two methods of examination ranges from 1.6% to 8.1% with a mean difference of 4.8% (Table 6A, Column 7). Thus in this

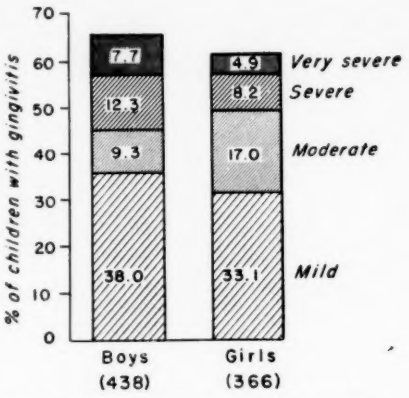


Fig. 4. Histogram showing distribution of different degrees of gingivitis in boys and girls (5-14 years of age).

group 95.2% of the children with gingivitis could be detected by examination of the anterior segments alone. In only 4.8% of the children with gingivitis, did the gingivitis occur in the posterior segments alone where it might have been missed if only the anterior segments had been examined. These facts indicate that by the examination of the anterior segments alone, the margin of error is increased by only 4.8%. On the other hand, the amount of time saved by this procedure may be as high as 75% since two segments are examined instead of six segments, and the segments examined are very readily visible.

Average P-M-A. If the average number of papillae and margins affected by gingivitis are computed from the examination of the ten anterior gingival units alone and these figures compared with those obtained by the examination of the entire mouth, only a very slight difference between the two sets of figures is demonstrable (Table 6A, compare Columns 4 and 6). If these figures are graphed, a remarkable parallelism between the two curves exists from ages 5 to 14 years (Fig. 6).

From the figures presented in Table 6B, it is apparent that 80% of the papillae and 85% of the gingival margins affected were located in the anterior segments.

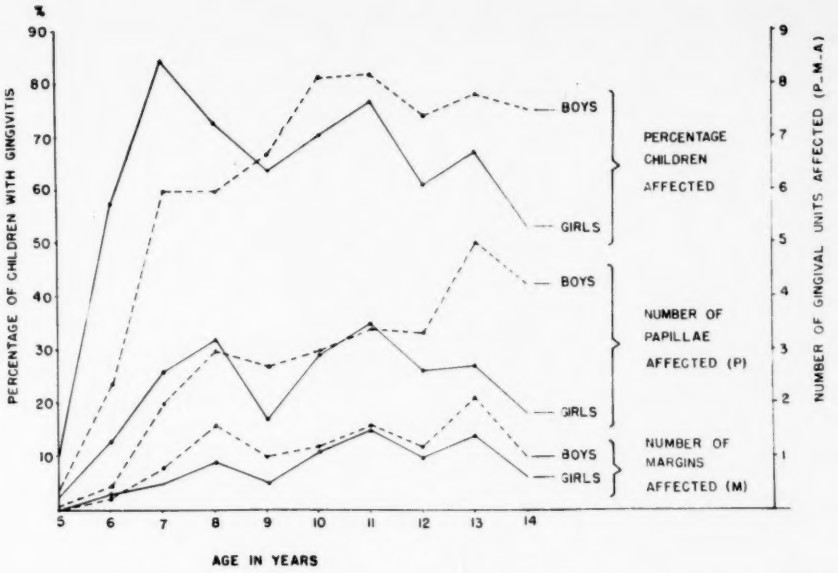


Fig. 5. Graph comparing gingivitis experience at different ages in 438 boys and in 366 girls of Melrose Park and Mooseheart.

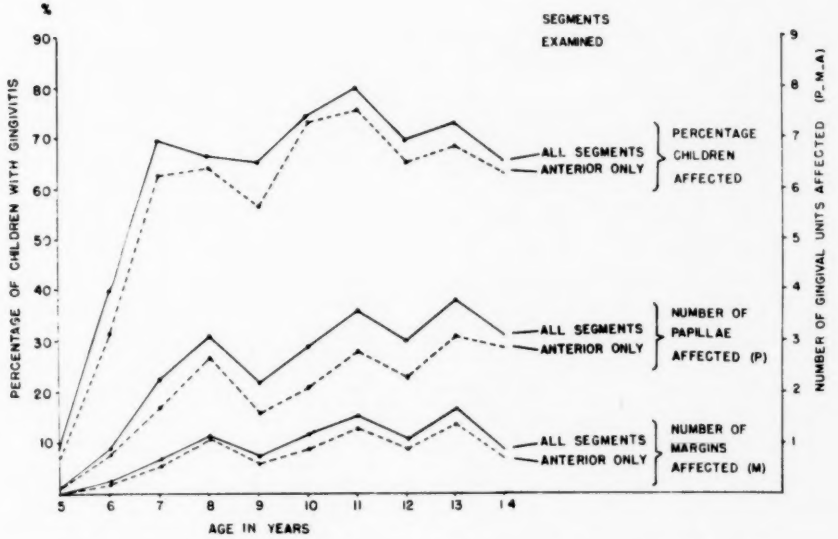


Fig. 6. Graph comparing gingivitis experience in all segments and anterior segments only.

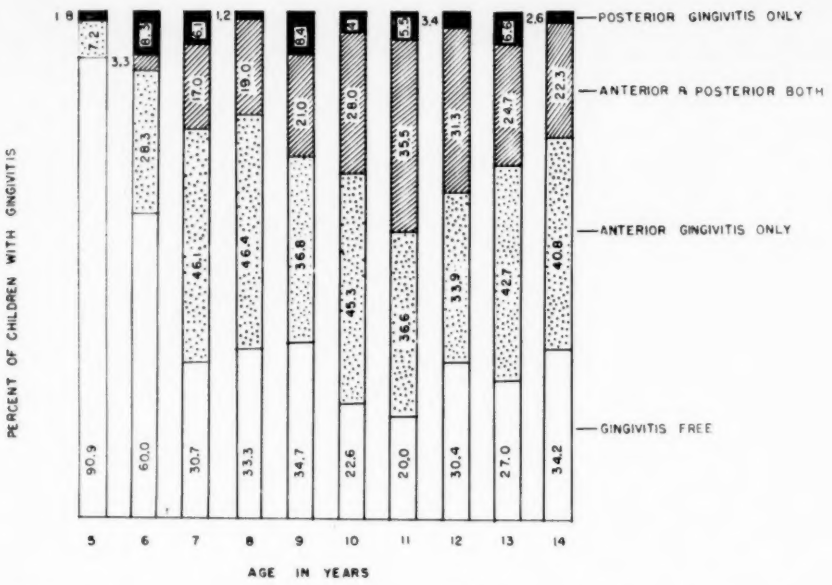


Fig. 7. Histogram comparing gingivitis experience in different segments of the mouth (804 children) 5-14 years of age.

Table 6A Column 8 also gives the difference between the Anterior and Total P-M-A. This is equivalent to the P-M-A of the four *posterior* segments. The figures show clearly the very small amount of gingivitis to be found in the four posterior segments. This was in spite of the fact that there are ten anterior units as compared with twelve to sixteen posterior units. The ten anterior gingival units exhibit 80% to 85% of the total gingivitis experience while twelve to sixteen posterior units *exhibit only 15% to 20% of the total gingivitis experience.*

It is also striking to note the great difference between the number of gingival units affected in the anterior and posterior segments. The anterior segments show a much greater number of units affected (2.14 papillae and 0.85 margins) than do the posterior segments (0.53 papillae and 0.15 margins). (Table 6A, compare Columns 6 and 8.)

DISTRIBUTION OF CHILDREN SHOWING GINGIVITIS IN THE ANTERIOR AND POSTERIOR SEGMENTS OF THE MOUTH

In order to further establish the validity of the use of the anterior segments alone as an index to the gingivitis experience of the entire mouth, it would be necessary to determine exactly how many children confine *all* their gingivitis experience to the anterior segments alone, how many children show gingivitis in the posterior segments alone and how many exhibit gingivitis in both areas or in none at all. Table 7 and Figure 7 present the analysis of our data towards this end.

From Table 7 it can be seen that 35.7% of all the children have no gingivitis at all and that an equal number (37.4%) have the gingivitis confined to the anterior segments alone. *Only 4.8% show gingivitis of the posterior segments alone,* the remaining 22.1%

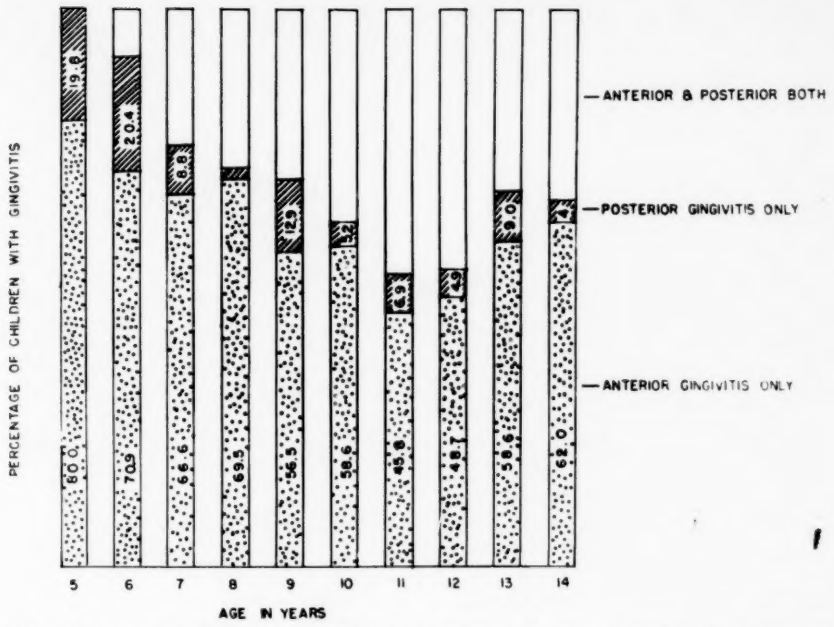


Fig. 8. Histogram comparing the percentage affected in anterior segments only and posterior segments only out of the total percentage of children affected.

showing gingivitis in both the anterior and posterior segments.

If this distribution is recalculated on the basis of children affected with gingivitis, it can be seen that more than half (58.3%) of the children with gingivitis confine the gingivitis to the anterior segments alone (Table 8). *Only 7.5% of the children with gingivitis confined that gingivitis to the posterior segments alone.* The remaining 34.2% showed gingivitis in both anterior and posterior areas.

These figures emphasize again (and prove statistically) the validity of using the anterior segments alone as an index of gingivitis experience in the entire mouth. If only the anterior segments are examined, 92.5% of the children with gingivitis will be discovered (58.3% anterior gingivitis plus 34.2% anterior and posterior gingivitis), and only 7.5% of the children who have gingivitis confined to the posterior segments alone will be missed.

Stated in another way, if we count all children examined including those with no gingivitis, only 4.8% of the total population will have gingivitis confined to the posterior segments. If we examine the anterior segments only, only these will be counted as gingivitis free instead of with gingivitis, an error of only 4.8%. The time and effort saved by examining only the anterior segments of large groups of children more than compensate for the small error.

The facts as stated for the entire group of all ages stand up with rather striking constancy *at all age* levels from 5 to 14 years. (Tables 7 and 8; figures 7 and 8.) These tables and figures show quite clearly that at no age level does the percentage of children with gingivitis confined to the posterior segments alone rise above 8.4% of the total

number of children examined or high enough to cause significant interference with the final result in a large group of children. Eight and four-tenths per cent is well within the margin of error in observation. At no time after the age of 6 years were there more than 12.9% of the children with gingivitis who had this gingivitis confined to the posterior segments alone. (At ages 5 and 6 the number of children with gingivitis is so small that the "unusual" case accounts for the high figures of 20.0% and 20.8% shown in Table 8.) The black areas in Fig. 7 (proportion of children with gingivitis confined to the posterior segments only) indicate the margin of error that might be introduced by examining the anterior segments only, rather than the entire mouth.

DISCUSSION

This study was undertaken with two primary objectives: (1) to apply the methods of assessing gingival disease described by Schour and Massler (1948) as the P-M-A Index, to a group of normal, healthy grade-school children—and in so doing to test its value as an epidemiologic method of assessing gingival disease in children and (2) to establish a "normal" quantitative baseline of values for the occurrence of gingivitis in school children with which other studies could be compared. The latter was a particularly sought objective since the children chosen for this baseline study were above the so-called "average" in terms of diet and nutritional status and enjoyed better than average dental care. In other words, these children came as close to an ideally nurtured and dentally cared for group of children as we could reasonably expect to find. For these reasons, we feel that the figures obtained may in reality be considered in terms of a baseline for future studies.

While many types of gingival diseases have been described, most of these have been classified under two major divisions—the inflammatory disease (gingivitis) and the degenerative diseases (Orban, 1947). Schour and Massler (1947) pointed out that the inflammatory changes are common in childhood while the degenerative changes become more apparent in the young adult after 25 years of age. In this study, gingivitis alone was assessed since other periodontal disorders were very rarely encountered.

Incidence of Gingivitis. A comparison of our data on the percentage of children with gingivitis with those in the literature indicates that our figures compare fairly well with those obtained by Ainsworth and Young (1925) in England and Wales on children aged 2 to 14 years; middle class Indian children (average age 13.8 years) as observed by Day (1940); and English children 11 to 14 years old as analyzed by King and Franklyn (1944) and King (1945). These studies showed that approximately 40% to 70% of the children were affected by gingivitis.

Similarly, the distribution of children into categories of mild, moderate or severe gingivitis as obtained by our method agrees fairly well with similar distributions as reported for healthy children in England and India. However, our figures do not agree at all with the figures on American children as reported by Messner et al. (1933-1934) or by Bruckner (1943) who indicate less than 10% affected. It is likely that the criteria employed in these two studies were such that inflammation of the gingiva was not classified as gingivitis unless it was relatively severe.

Effect of Malnutrition on the Occurrence and Severity of Gingivitis. The studies by Day (1940, 1944) and Day and Shourie (1947) in India; King and Francklyn (1944) and King (1945) in England; and by Schour and Massler (1947) in Italy all indicate that the occurrence and severity of gingivitis tends to increase with an increase in malnutrition. This is indirectly supported by the findings in this study of a lower prevalence rate and lower severity of gingivitis in this well-nourished group than in the malnourished groups reported by the above authors. However, a specific study of occurrence of gingivitis using the P-M-A index on a group

of malnourished children should be done for statistical accuracy.

Age Incidence. Our study showed that the percentage of children affected with gingivitis increased with increasing age. It increased rapidly from 5 and 7 years (from 10% to 70% affected). Thereafter it plateaued but remained high, up to the age of 14 years (65% affected). Similar trends were observed by Ainsworth and Young (1925), Messner et al. (1933-1934), Day (1940) and Schour and Massler (1947). The increase was found not only in the number of children affected, but also in the severity of gingivitis (Table 3).

It was indicated by Schour and Massler (1947) that for the most part inflammatory lesions of the gingiva tend to affect first the gingival papilla. From the papilla the inflammatory lesions spread to the marginal gingiva. In other words, the severity of the disease has increased with the involvement of marginal gingiva. If the inflammation becomes more severe, it progresses towards and involves the attached gingiva. This study confirms that observation.

Eruption Gingivitis. In our examination the clinical signs of gingival disease were recorded around both the erupting and fully erupted tooth. It was observed that the character of the gingivitis was different in the group 5 and 7 years of age from that at other age levels. During this age period of 5 to 7 years, a large number of teeth are erupting into the oral cavity. The sharp rise in gingivitis left us with the impression that it was caused primarily by the eruption of the four incisors and the first permanent molars. The percentage of children affected remained high until the age of 8 years, and thereafter dropped slightly at the age of 9 years. A quiet period, when few teeth are erupting, occurs between 8 and 9 years. Beginning with the ninth year another rise is seen in the occurrence of gingivitis. The increase was slower and was probably related to the slower eruption of the bicuspid teeth. The occurrence of gingivitis remained high from 10 to 12 years of age probably because the bicuspid erupt over a longer period of time. From the above considerations, we feel that the inflammation during this age period may be regarded as "Eruption Gingivitis."

Sex Difference in the Incidence of Gingivitis. While there were significant differences between the gingivitis experience in boys and in girls, such differences were not very striking, nor consistent.

In the age group from 5 to 8 years, girls in general showed a higher percentage affected by gingivitis than did the boys. The difference might well be explained on the basis of an earlier eruption of teeth in girls.

From 9 to 10 years of age no differences in gingivitis experience could be determined in boys and girls. However, from 10 to 14 years of age, there was a tendency for boys to show more gingivitis than girls. This might be explained on the basis of better oral hygiene in girls. Girls tend to become more conscious of cleanliness as they approach puberty than do boys.

Validity of Examining the Anterior Segments Only. An attempt was made to establish the validity of the use of the anterior segments alone as an index to the gingivitis experience of the entire mouth. In order to test this hypothesis, different segments of the mouth were analyzed separately. It was observed that if only the anterior segments were examined and recorded, approximately 92% of the children with gingivitis would be detected, and approximately 82% to 85% of the total number of papillary and marginal gingivae affected by inflammation would be recorded.

It is clear that for purposes of comparison between large groups, examination of the anterior segments alone is adequate. The time saved would allow the profitable examination of much larger numbers of persons.

SUMMARY AND CONCLUSIONS

This study was undertaken for the purpose of assessing gingival disease by applying the method of P-M-A Index to a group of 804 normal, healthy grade-school children living

in Chicago suburbs (Mooseheart and Melrose Park). From this study, it was hoped that we would establish a "normal" quantitative baseline of values for the occurrence of gingivitis with which other studies could be compared.

Each child was examined with a tongue blade under good artificial light. Gingival tissue of only the buccal and the labial surfaces was examined.

A semi-quantitative method proposed by King (1945) was refined toward more exact quantitative and qualitative evaluation of the gingival disease. According to this method (P-M-A Index), the gingiva is divided into gingival units consisting of the interdental papilla (P) the marginal gingiva (M) and the attached gingiva (A). See figure 2A.

Since inflammatory lesions tend, for the most part, to begin in the papillary gingiva and to spread into the marginal gingiva and then to the attached gingiva in severe gingivitis, the degree or severity of the inflammation can be indicated by its location: mild gingivitis being confined to the papillae; moderate gingivitis involving the marginal gingivae; and severe gingivitis affecting the attached gingivae. It, therefore, was logical to examine first the papilla (P), then the labial marginal gingiva (M), and last, the attached gingiva (a) and to record any deviation from normality in those areas.

It was found that:

1. The percentage of children affected with gingivitis increased significantly with increasing age. Such increase was found not only in the number of children affected, but also in the severity of the gingivitis (Table 3 and Figs. 3A and 3B).

2. The character of the inflammation observed at the time of eruption of the teeth led us to use the term "eruption gingivitis." It was felt that the eruption of the teeth accounted for the sharp rise in gingivitis from 5 to 7 years of age.

3. More girls were affected by gingivitis from 5 to 8 years of age, while more boys were affected from 9 to 14 years.

4. The average P-M-A Index or average number of papillary, marginal and attached gingival units affected per child at the different age levels is given in Table 3.

5. Table 3 shows that the average child between 5 and 14 years has 2.7 papillae and 1.0 margins affected by gingivitis. Attached gingivitis (periodontitis) was practically non-existent in our group. Approximately 36% of the children were free of any gingivitis, while another 36% suffered from only mild gingivitis (less than average P-M-A). Approximately 13% showed a moderate amount of gingivitis (3–2–0 to 5–2–0); while 15.7% showed a severe or very severe gingivitis (6–2–0 or more).

6. Our study offers statistical proof that the examination of the anterior segments alone for the occurrence and degree of gingivitis is a valid index of gingivitis experience of the entire mouth.

ACKNOWLEDGEMENTS

This investigation was supported by a research grant from the Division of Research Grants and Fellowships of the National Institute of Health, United States Public Health Service.

Grateful acknowledgements are made to the following persons:

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John M. Williams, D.D.S., Chief Dentist of Mooseheart, Illinois.

Arnold C. Erxleben, Principal of the Melrose Park Lutheran School.

Paul A. Topel, D.D.S., Examining Dentist of the Melrose Park Lutheran School.

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3. Day, C. D. M., and Tandan, G. C., The Incidence of Dental Caries in the Punjab. Brit. Dent. Jnl. **69**:381-389 (Dec.) 1940.

(Concluded on page 196)

Preliminary Program

Thirty-Fourth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

October 26, 27, and 28, 1950

Atlantic City, N. J.

THURSDAY, OCTOBER 26, 1950

11:00-12:00 a.m.

A Mid-Century Appraisal of Developments in Periodontology—Harold J. Leonard

2:00-2:30 p.m.

The Structure of Normal Gingivae—Balint Orban

2:30-2:50 p.m.

A Case of Periodontitis—A Bio-Mechanical Approach to Treatment (Case Report)—Lewis Fox

3:00-3:45 p.m.

Further Studies in the Histologic Changes in the Gingival Tissues in Health and Disease—Joseph Bernier

3:50-4:10 p.m.

Was Gingivectomy Necessary? (Case Report)—Edward Ball

4:15-5:00 p.m.

Changes in the Epithelial and Connective Tissue in Periodontal Disease—Paul Boyle

5:00-5:20 p.m.

Various Phases in the Treatment of a Case of Periodontal Disease (Case Report)—Henry Goldman

FRIDAY, OCTOBER 27, 1950

9:30-10:15 a.m.

A Basic Approach to the Problem of Bone Loss in Periodontal Disease—Irving Glickman

10:20-10:40 a.m.

Periodontosis in a Child, Resulting in Exfoliation of the Teeth (Case Report)—S. Leonard Rosenthal

10:45-11:30 a.m.

The Bacteriologic Aspect of Periodontal Disease—Ned Williams

11:35-12:00 a.m.

A Radiographic and Histologic Study of Reattachment—A Case History—Frank Beube

2:30-3:15 p.m.

Occlusal Dynamics—Lewis Fox

3:15-4:00 p.m.

Muscular Force, Occlusion and Function as Etiologic Factors In Periodontal Disease—Max Gratzinger

4:10-4:30 p.m.

Ligating in Periodontia Practice—Leonard Hirschfeld

4:30-5:00 p.m.

Principles of Therapy Illustrated by Case Reports—Harry Lyons

SATURDAY, OCTOBER 28, 1950

9:30-11:30 a.m.

Round Table Discussion—"The Treatment of Periodontal Pockets"—Henry Goldman Moderator; Irving Glickman, Harry Lyons, Harold Ray, William G. McIntosh

Editorials

Change in Emphasis Needed

TO THE objective observer, unhampered by the narcosis of custom, the American Dental scene presents an inexplicable paradox. The paradox is highlighted by an inconsistency between the nature of the dental problem of the population, the teaching program designed to prepare dentists to cope with this problem, and the treatment planning of most dentists. On the one hand, the observer learns that beginning with the fourth decade of life, periodontal disease is the greatest single cause of tooth loss.[1,2] In addition, because of a steadily increasing life expectancy, each year finds a large proportion of the population at an age level at which tooth loss is more likely to result from periodontal disease than caries.[3] On the other hand, he finds the following regarding the teaching of periodontology.

(a) It occupies only 2.10% of the time of the entire dental curriculum.[4]

(b) The time allotted to dental materials exceeds that devoted to periodontology.[4]

(c) Prosthodontia and operative dentistry combined receive 22.1 times the attention given to periodontology.[4]

(d) In evaluation of the quality of clinical teaching periodontology rated 11th on a list of 16 clinical subjects, inferior to dental ethics but on the same plane as jurisprudence.
Prosthodontia, oral surgery, and operative dentistry occupy the first, second, and fifth positions respectively in this quality evaluation.[5]

(e) In a total of 38 schools, the teaching of periodontology is rated as follows: excellent and good—10; fair and poor—28. The clinical teaching of prosthodontia is rated: excellent and good—30; fair and poor—8; oral surgery: excellent and good—23; fair and poor—15; operative dentistry; excellent and good—19; fair and poor—19.[5]

If the observer were not previously conditioned by an appreciation of the comprehensive objectives of present-day dental education, he might arrive at the following erroneous conclusions from the aforementioned facts:

Abstracted from a paper by Dr. Irving Glickman, of Tufts College Dental School, and read before the Periodontia Section of the American Association of Dental Schools, March 28, 1950.

1. Brekhus, P. S., Dental Disease and Its Relation to the Loss of Human Teeth. J.A.D.A. **16**:2247, 1929.

2. Allen, E. F., Statistical Study of the Primary Causes of Extractions. J.D.R. **23**:453, 1944.

3. O'Rourke, J. T., Dental Care for Children in Relation to Geriatrics. J.A.D.A. **34**:595, 1947.

4. "Dental Education Today," Harlan Hoyt Horner, 1946, University of Chicago Press., p. 235.

5. Idem, p. 246.

(a) That dentists are somewhat oblivious to the existence of periodontal disease.

(b) That dentists are more concerned with repairing and replacing teeth than the prevention of their loss.

(c) That dentists do not realize that the more expertly the ravages of dental caries are repaired, the more teeth will be present in people when they reach the so-called "periodontal susceptible age."

The significance of periodontal disease as one of two major causes of tooth loss and the important role of periodontology in dentistry should be clarified for the dental student at the outset of his career in the dental school. He should be made aware of the fact that his obligations as a dentist will not be essentially confining to filling and replacing teeth. It should be made clear to him that because of the prevalence of periodontal disease, care of the supporting tissues is part of "bread and butter" dentistry which he should expect to manage in his practice with the same ease and confidence as filling a tooth or taking an impression. He should be made to understand that his obligation to acquire proficiency in the periodontal field is just as much a prerequisite for graduation as the other major clinical requirements in the curriculum.

Dr. Gottlieb

In the passing of Doctor Bernhard Gottlieb the dental profession and the scientific world in general have lost a fellow worker of monumental achievement and versatile interests.

Doctor Gottlieb's participation as an investigator in the field of dentistry covers many areas. He will be remembered for his profoundly influential findings in connection with the eruption of the teeth and the formation of the epithelial attachment. His publications deal with many aspects of periodontal disease and, in his latter years he extended his interests to include the histopathology of dental caries and its clinical control. While Doctor Gottlieb's concepts and conclusions have aroused both acclaim and controversy within the profession, the impact of his imaginative and daring intellect and the profundity of his experience have constituted a stimulus to dental investigators for nearly three decades; and are attested by the constant references to his findings noted in current literature.

A multitude of his colleagues from every quarter of the globe will be acutely conscious of the loss occasioned by his death and those who have been privileged to know him personally will continue to pay tribute to his qualities of mind and of heart.—R.G.A.

The Etiology of Periodontal Disease: A Review of Current Literature

JOHN H. MOSTELLER, D.D.S
Mobile, Ala.

AFTER years of clinical observation and comprehensive research the etiology and pathogenesis of periodontal disease remain obscure. Before a rational approach to treatment can be made a thorough investigation of possible as well as proven etiological factors must be undertaken. Temporary alleviation of the condition may be obtained by an empirical treatment of symptomology but more lasting benefits are derived only by a therapeutic regime aimed at the causative agents. A correct evaluation of any lauded treatment procedure can not be made from immediate clinical appearances only, but must be through a correlation of the therapy with the pathogenesis of the disease.

Periodontal disease, it is agreed, is best treated in its incipiency, but again, this success may be due to less severe symptoms which return to the clinical appearance of health more easily rather than actual removal of the etiology of the disease. Temporary expedience may impress dramatically both patient and operator but a genuine service in periodontia is rendered only when the future plight of the tissues is considered as strongly as the present complexion of the condition.

Categorically the etiology of periodontitis has been divided into those factors of intrinsic and extrinsic or systemic and local origin. Opinion as to the role played by known or unknown general conditions varies from an estimation that systemic disturbances motivate ten per cent of all cases that confront the periodontist[1,2] to an insistence that the entire problem is a systemic one.[3] Another estimation, shared by several, is that fifteen per cent of all cases are caused by factors other than local.[4,5] Most popular is the conception that while subtle systemic embarrassment may predispose periodontal disease, with the exception of a few well defined maladies, local influence is necessary.[6,7,8,9] Some feel that basically extrinsic factors are at fault, with a few recognized intrinsic disturbances contributing at times, in all forms of periodontoclasia except the believed degenerative type designated as periodontosis by the Committee on Nomenclature of the American Academy of Periodontology.[10,11,12] They feel that this difficult to manage pattern of periodontal disease is the result of some vague general disorder with the local principles occupying the contributory position.

Systemic irregularities most commonly accused as the etiology of periodontoclasia are nutritional deficiencies, hormonal imbalance, blood dyscrasias, diabetes mellitus, drug poisons, and psychosomatic manifestations.

NUTRITION

Unquestionably, an acute avitaminosis C results in scurvy with gross destruction of the alveolar bone and periodontal membrane which leads to the loss of the teeth; but a subscorbutic deficiency does not appear to exert such a strong influence on the periodontium without concomitant local agitation in humans. *Boyle*[13] has been able to demonstrate destructive changes in proportion to the severity of the vitamin C privation in tests on guinea pigs. These changes, such as loosening of the teeth, hemorrhage of the gums, and resorption of the alveolar bone, ceased and there was rapid repair when ascorbic acid was added to the diet. Although there is a marked similarity between the periodontal apparatus of the guinea pig and man, acute signs of a vitamin C deficiency appear only after a much longer period of depletion in the latter. *Boyle*[14] does report minimal tendencies toward gingival hemorrhage in man with a deficiency of moderate duration.

Burrill,[15] in studies of blood plasma vita-

min C levels, concluded that avitaminosis C does not consistently disclose itself in the oral cavity of man. *Ungley and Harton*[16] also failed to find any increased periodontal symptoms in sailors with marked deficiencies of vitamin C over those of a control group saturated with the vitamin. While many authors[6, 7, 8] warn of periodontal distress from an exigency of ascorbic acid, it appears, from the evidence based on research rather than opinion, that a vitamin C deficiency, unless of extreme duration and severity thus presenting a true picture of scurvy, can be considered only as a predisposing factor.

Glickman, Morse, and Robinson[17] reported greater alveolar bone loss, after packing amalgam in the interproximal spaces, in albino rats on starvation diets than in those receiving normal adequate rations; but there was bone resorption in both groups. They further found some bone loss in a few cases on the starvation diet without the added insult of the interproximally packed amalgam, but no destruction in those on the adequate diets and free from this interproximal irritation. The condition simulated here is of the most extreme nutritional insufficiency, rather than just one of ascorbic acid incompetence, and yet the reflection in the mouth is slight when appraised as a clinical entity. It must be considered, however, as a definite contributing factor.

In another study, *Boyle*[14] reports that while proper intake of vitamins A and D were essential to the formation of bone, a deficiency of either or both in guinea pigs did not affect the health of the periodontal tissues. Neither could he find changes in these structures when the experimental animals were placed on starvation diets. Vitamin B deficiency has been listed as a possible cause for periodontal pathology,[1, 7, 8] but there is no evidence to substantiate this. We must index nutritional deficiencies only as a contributing or predisposing factor in our atlas of the etiology of periodontal disease.

Leonard,[8] in his mensuration of the nutritional components, surmises that the lack of self-cleansing effect of the typical modern diet is more important from a causative standpoint than supplementary food deficiencies. Since cementum deposition seems to be associated with a continuous passive eurption of the teeth, the modern non-abrasive diet may likely play another paramount role in its failure to wear down the teeth and thus stimulate this continuous eruption.[18] Likewise, today's soft diet reduces the atrophy of the gingival crest which must stay in balance with the epithelial proliferation at the apical end of the gingival crevice if we are to maintain a fairly constant depth to this crevice.[19]

HORMONAL INFLUENCE

While the endocrine glands are in a large way responsible for the growth and development of the jaws,[20] no proof, clinical or otherwise, has ever been presented to link dental disease with their dysfunction.[21] Cases have been reported in the literature of improvement of gingivitis with the application of sex hormone ointments,[22] but others have met failure with such therapy although the patient associated acute exacerbations of the gingiva paroxysmal with the menses.[23] Hormone imbalance during pregnancy has long received the blame for both gingival and periodontal troubles,[1, 6] however, extensive investigation has proven that "pregnancy gingivitis" does not exist as an entity.[24, 25, 26] Gingival reaction to local irritation seems to follow a definite trend toward proliferation during gestation and the hormonal situation may possibly be responsible for this exaggeration of symptoms, but the local irritation must be present in order to have any gingival reaction. Pregnancy should be acquitted of its blame for periodontitis in the same manner it has been vindicated from the false accusations of laymen, physicians, and even dentists of causing dental caries.

Addison's disease, which is the result of an impairment in function of the adrenal cortex, manifests itself in the oral cavity by melanotic pigmentation; and hypothyroidism often by "epulus type" growths; but these signs serve as diagnostic warnings only and are not directly related to periodontal disease.[27]

BLOOD DYSCRASIAS

Most articles itemizing the etiological factors in periodontal disease include blood dyscrasias with special reference to leukemia. Cook[28] gives a complete histological as well as clinical description of the gingiva in acute leukemia, illustrating one case where the hyperplastic gum tissue completely covered the dentition. There is no doubt that this condition is caused by the blood dyscrasia as in those cases that become manageable the mouth returns to normal simultaneously with the improvement of the hemopoietic disturbance. Periodontal distress is indeed insignificant to a patient suffering with an almost totally fatal acute leukemia and therefore again the condition of the mouth serves only as a diagnostic warning of the systemic malignancy. One might immediately suspect the implications of this disease in periodontitis should the blood dyscrasia remain in a chronic state. Once more if we properly evaluate the literature, we learn that a chronic leukemia does not reveal itself by changes in the periodontium.[25]

Most clinicians have observed the glossitis associated with anemia but no significant changes in either the gingival or periodontal tissues have been noted.[27] Thrombocytopenia purpura sometimes, but not always, exhibits itself in the oral cavity by the formation of purpura and echymosed areas but these lesions are more frequently found on the buccal mucosa and lips than on the gums.[9] When the gums are involved, there is a proclivity for hemorrhage but no specific alteration of either the alveolar bone or the periodontal membrane. This blood abnormality is usually associated with general debility of consequential severity and to reiterate, the oral manifestations must be inventoried as diagnostic only. The comparison of blood studies of a large number of patients suffering from periodontoclasia with those of an equally large control group indicated no significance to slight blood dyscrasias.[29]

DIABETES MELLITUS

Diabetes is considered an almost insurmountable obstacle by some men practicing periodontics, but it is difficult to discern upon what reasoning they predicate this defeatism. A symposium[25] of most equitable histopathologists agreed that bone destruction did occur, in some cases, in proportion to the severity of the unchecked diabetic condition, but since the patient's very life hinges upon his conscientious observance of a restricted diet and/or insulin supplementation, the periodontal condition of an acute diabetic becomes almost academic. Diabetics under treatment presented no specific periodontal pathology. *Glickman*[30] discovered in tests with alloxan induced diabetic albino rats that a non specific destruction of bone occurred in only thirty-nine per cent of the cases. Our conclusion from these studies can only be that we must certainly not expect periodontal difficulties in all diabetic patients, and neither can we expect resolution of such a condition, if it exist, by the mere regulation of the pancreatic insufficiency. While we must not overemphasize the importance of diabetes mellitus and sit back and wait for the physician to "pull the stroke oar," it is our responsibility to insist on a blood sugar determination and urinalysis in cases which do not offer sufficient local etiology. In any such case recognized as diabetes, the indebtedness of the patient will extend far beyond his appreciation of our endeavor to save his teeth. Even when the prognosis is such that extraction is indicated, the patient should certainly be referred to a competent internist for possible diabetic inclinations.

DRUG POISONS

Sensitivity of some patients to specific drugs has been observed by the profession for years.[6, 27] This reaction can not be explained by the pharmacology of the medicinal but assumes the appearance of an allergy. Where the drug is selectively prescribed, substitutes can be given with a disappearance of the gingival condition. Where the drug is specific, the only alternative is to determine the minimum effective dosage. If after the ascertainment of above, the gingival condition persists, treatment can be aimed only at symptomology which is not

only unscientific but usually discouraging due to the flaccidity of the results obtained.

PSYCHOSOMATICS

Psychosomatic medicine is a relatively new and to some an unproven science. With such an infancy in medicine, it is not difficult to understand why it has such an undetermined position in dentistry. There are those who report correction of gingivitis by psychological rehabilitation when other methods failed.[31, 32] While there is nothing in the literature to generally apply their contention, neither is there anything to contest it. This phase of possible eitology is patent for further investigation.

It is not necessary to dwell in the vagueness of psychosomatics to correlate bruxism with nervous instability.[31, 33] It is, however, necessary to accept the observations of many as to the pathogenesis of periodontal disease from traumatic forces in order to place any notability on this nervous habit.[34] It is interesting to note that one investigator disclaims all local factors in one thesis,[3, 35] and then warns of excessive gum chewing or nervous grinding and gritting of the teeth in another.[36]

GERODONTOLOGY

Another consideration under general etiology must be the aging process. Thomas[37] found some softening of the periodontal fibers and moderate resorption of the interdental septum in the aged but concluded that the supporting structures can remain in good health provided the patient is free from all local and systemic causative elements. Although the gingival crevice does shift apically with age the teeth nevertheless may remain firm in their sockets and provide complete masticatory service.[38] This can be explained by studying the work of Goldman.[19] As the teeth are worn down with advancing age there is a continuous passive eruption of the teeth with synchronized epithelial proliferation at the apical base of the gingival crevice. This apical movement of the floor of the gingival crevice should be associated with atrophy of the gingival crest and thus maintain a fairly constant depth to the crevice. However, if the latter process is unable to remain in step with the former there appears to be little harm done in a healthy mouth, for Goldman, in another study,[39] was unable to determine any correlation between the depth of the gingival crevice and the attachment of the periodontal membrane or the degree of resorption of the alveolar bone.

HISTOPATHOLOGY

Gottlieb attributes the entire periodontal problem to systemic causes because of his theory that the condition of the cementum, rather than the periodontal membrane, determines whether or not there will be a barrier to the down growth of the epithelium.[3, 35] He believes that if the outer layer of the cementum at the base of the crevice is uncalcified and thus possessive of a high degree of vitality there will be continual deposition of cementum, upon which it is agreed attachment is dependent. In turn, if this outer coat is an old calcified layer, no deposition is possible and there will be a progressive invagination of the epithelium. Gottlieb postulates that the condition of this outer cementum layer is regulated by some unknown systemic influence. Since local factors affect the condition of the fibers of the periodontal membrane and not that of the outer coat of the cementum, he discredits them from having anything to do with periodontitis. He likewise charges that a change in function, demobilization, or traumatic occlusion will not alter the deposition of cementum in a mouth with a vital uncalcified outer layer.

From a clinical standpoint, many men, including the author, can not accept the theory advanced by Gottlieb. Other equally qualified histopathologists interpret the microscopic findings in a different light. *Lefkowitz*[18] attributes the deposition of cementum to the periodontal membrane and has shown that formation may take place regardless of the qualities possessed by the calcified connective tissue covering the root surface. If an uncalcified area is absent after repair

of resorbed cementum the periodontal membrane can nevertheless be stimulated to its function of cementum formation provided the irritating cause of resorption is removed. *Goldman*[39] presents histological evidence that the healthy fibers of the periodontal membrane prevent epithelial penetration, but if an injured space is created, the invasion will take place until the cause of the injury is removed. He shows definite evidence of cementum repair after removal of the irritation provided the periodontal membrane is still intact to function in cementum formation. It is therefore possible to obtain a reattachment, which is in reality a new attachment as pointed out by Orban, in what Goldman deems a true periodontal or bony pocket. Such a pocket is made up of the root surface on one side and an alveolar bone matrix on the other with the injured membrane and invaginated epithelium occupying the space between.

In the broad pocket, which is made up of the root surface on one side and epithelial tissue on the other and does not extend below the crest of the alveolar bone, there is no scientific basis for a claim of a true reattachment.[39] It is possible to obtain a functional close adaptation by restoring the tissues to health and some men have referred to this as a reattachment,[40] claiming that the epithelial tissue lining of the pocket differentiated into a connective tissue layer and attached itself to the root surface of the tooth. It is impossible to have attachment of fibers without cementum deposition and there is no indication of this formation without the stimulation of the periodontal membrane. It is however possible and desirable to obtain reattachment and cementum repair at the base of the gingival crevice in these pockets.[41]

INFECTION

The significance of bacteriological findings in periodontal disease has received debate for years, and the opinions expressed in the recent literature are still at odds. Box[42] accepts the fact that extrinsic factors are immediately responsible for periodontoclasia, but he believes that intrinsic mechanisms set up a predisposition. He explains the destruction as being caused by toxins which he guesses are produced by a fungus found in the gingival crevice. Fish, in his text, proposes a toxin with the conjuncture that they are liberated by bacteria. Other investigators[43] recognize the suggestion of toxins by the presence of necrotic foci in the deeper tissue and speculate that they are produced by bacteria and/or degenerating cells in the superficial ulcerations of the gingiva. They do not however attempt to imagine that some mysterious systemic mechanism provides for the culturing of these organisms, but assume that tissue damaged by some local cause provides the media.

Rosebury[44] insists that infection is an indispensable factor in all the forms of periodontal disease. He feels that the reported benefits from penicillin are conclusive evidence of this. Any benefits from antibiotics are short lived at best and therefore we are compelled to suspect that infection is only a secondary phenomena. It may well be responsible for many of the destructive changes in the supporting tissues of the teeth, but this has not been proven. Rosebury believes that the infection is caused by organisms that are harmless in the presence of healthy tissue but are capable of propagating and invading tissue injured by precursory changes. He attributes these precursory changes to both local and systemic origin and declares that the severity of the disease is regulated by the nature and degree of these initial insults to the tissue.

Bernier[45] demonstrated very clearly that the histopathological process in periodontal disease is a nonspecific exudative inflammation with either resolution and repair or necrosis being the termination according to the severity and duration of the causative factors. This is the picture he found even in so called periodontosis. Histological differential diagnosis is therefore based upon the degree of destruction rather than the character of the process.

Williams[46] reports that he finds an absence of the inflammatory histopathological

picture in periodontosis or periodontitis complex, but rather focal necrotic areas which he attributes to enzymes liberated by the fungus colonies related to *Leptothrix falciformis* found in the gingival sulcus. He believes that this substance may extend to other tissues either by diffusion or by a lymphatic route. Williams suggests that when the necrotic areas are found with concomitant inflammation, the disease is a combination periodontitis complex-simplex. Two others report,[47] after extensive bacteriological studies, that they must conclude that, while bacteria are present in greater quantities and with greater virulence in mouths affected with periodontoclasia, these organisms seem to be secondary invaders only, determining if there will be a suppurative stage to the disease.

After judging the evidence upon which these investigators make their assertions, one can conclude that bacteria are not a primary etiological factor in periodontal disease but their increased number is more a result than a cause. Koch's postulates can not be fulfilled and neither is the severity of an already existing case of periodontoclasia intensified by innoculating the gums with organisms from a mouth suffering with more violent symptoms of infection. Bacteria can and probably do cause secondary infection and very possibly are guilty of serving as a foci of infection to other parts of the body, as suggested by Ray and Orban.[43]

The importance of the fungus colonies frequently found in the gingival sulcus has certainly not been established and there is need for extensive study of their morphology, the biochemistry of any toxins, and the pathogenesis of these toxins when identified, before practitioners can pay credence to this canal of thought. There is likewise need for proof that any particular bacteria are capable of producing destructive toxins in periodontal disease. Since the condition does resist any effort to establish it as an infectious process, the theory of toxin liberation by degenerating cells as a result of one or several causes seems to deserve further study.

The interpretation of the histopathological picture of periodontosis may seem confused but it is not difficult to suppose that the variances are due to the study of sections made at different stages of the disease. Microscopic studies revealing necrotic areas without marked inflammatory signs may well be the terminal stage of a cycle. The evidence seems to support the notion that the pathology of all periodontal disease, including periodontosis, is one of exudative inflammation in response to some causative irritation rather than one of general degeneration.

Since the periodontosis does not respond to oral prophylaxis as does periodontitis simplex, there is quite naturally greater opportunity for the inflammatory process to terminate in necrosis; and since in these cases the etiological factors seem to injure the periodontal membrane rather than the gingiva, there is no cause for gross cellular infiltration of this contiguous tissue. It is therefore obvious that the histopathological picture in such cases will be of less severe inflammatory nature. The evidence still remains that the histopathology is the same in all periodontoclasia, the only difference being the amount of inflammatory activity with associated resolution and repair versus the amount of terminating necrotic destruction. At this point it is well to remember that inflammation is a defensive process and the severity of the disease can not be ascertained by utilizing it as a criterion.

LOCAL FACTORS

In reviewing the local factors responsible for injury to the periodontium and subsequent periodontal disease, salivary calculus is the first to receive consideration by most authors. Although there was a long period, after Pierre Fauchard's first recorded observation of "pyorrhea" in 1746, that men attributed the malady solely to general causes and considered it a "kind of scurvy," clothed in mystery, the importance of removing calculus or "tartar" from the teeth soon became recognizable. That these deposits cause embarrassment to the gingival tissue is well established as is their agreed origin of precipitation from the saliva. Box believes "seru-

mal" or subgingival calculus, which is more notorious in its role in the etiology of periodontoclasia, has a distinctly different derivation.[48] He states these injurious formations on the necks and root surfaces of teeth are caused by a calcification of the inflammatory exudate resulting from some other primary local factor. One of the most common of these primary factors is the first mentioned salivary calculus, with bacteriological infection playing an intermediate part. Essentially, the contents of "serumal" calculus are materia alba (food debis), mucus (from saliva), dead epithelial cells, purulent matter (from anaerobic organisms), and other bacteria.[49] Since it has been definitely shown that subgingival deposits are a secondary rather than a primary result of poor oral hygiene or some other local irritation, the author can not see the rationale of those who recommend routine gingivectomy on the hypothesis that unless the pocket is removed, the patient can not keep clean the surface of the tooth covered by the epithelium. In reality, if the patient will maintain good supragingival hygiene and equally as important, if all other local factors are removed by the operator, there will be no cause for the formation of subgingival calculus. *Goldman*[39] believes there is nothing harmful about a deep gingival crevice.

OPERATIVE DENTISTRY

The patient is often justly indicted for his negligence of oral hygiene, but in many cases the dentist is due censoring for his lack of respect for the interproximal tissues as evidenced by his too frequent indiscriminate use of separators, ligatures, gingival wedges and etc.[50] He likewise is sometimes guilty of supineness in his administration of oral prophylaxis, the importance of which is ranked only slightly by that of the hygienic efforts of the patient.

The literature,[49, 50, 51] is replete with condemnation of men who promote periodontal disease through perfunctory operative technic. The injury to the periodontium that is primarily or secondarily the result of overhanging gingival margins, faulty interproximal contacts, uneven marginal ridges, improper occlusal topography and anatomical contour, deletion of sluice ways for the deflection of food, and other omissions from good operative dentistry is not of insignificance in the causation of pyorrhea in many mouths. It is not only the responsibility of the operator, dedicated to the conservation of dentition, to fabricate required restorations in harmony with the biological requirements of the supporting tissues, but also to insist on the replacement of existing faulty operative work that is unquestionably causing embarrassment to those structures.

ABNORMAL OCCLUSAL STRESS

Although interest in occlusal trauma as an etiological factor for the causation of periodontal disease has been most pronounced during the last decennium, the theory is almost fifty years old. *Karolyi*,[2] of Vienna, is credited with originating the idea in 1894 and *Bonwill* was one of the earliest advocates. *Stillman*, *McCall*, and *Box* have made well known contributions to the literature on this subject. Most recent essays on the treatment of periodontal disease include the importance of an equilibrated occlusion whether the essayist favors a surgical[52, 53] or so-called conservative mode of treatment.[6, 54, 55]

It is impossible to state that occlusal disharmony is the most important cause of periodontoclasia, but it is certainly one of the most frequently ignored. Most articles either dismiss the subject with the vague generality that traumatic occlusion should be adjusted, or else complicate the procedure through modified and inconsistent terminology to a point that unless the reader is well acquainted with equilibration, it is most difficult for him to gain any practical knowledge from the manuscript.[24]

It is obvious that the periodontal tissues need some stimulation by occlusal forces for all are acquainted with the consequences of allowing a tooth to remain in the arch without an antagonist. Since a tooth is suspended in a socket by minute fibril attachments, vertical forces serve this purpose of strengthening the periodontium through stimulation.

Even if this axial stress is excessive there is opportunity for increased pressure only at the apex, and if the tooth be multirooted at the bifurcation. *Breitner*[56] subjected monkey's teeth to continuous excessive vertical stress for three weeks and reported periodontal changes at the apex and bifurcation only, with the greatest change taking place at the latter since the stress was over a larger area of the membrane. Lateral stress, however, which caused pressure along a large area, resulted in resorption of the supporting bone with a greater loss occurring on the side of pressure than on the side of pull. This may explain why so often there is more bone destruction on the buccal of the upper molar roots and the lingual of the lowers, since interference in the lateral working masticatory stroke usually exerts force in this direction. By the angle of inclination of the teeth in relation to a vertical plane, the uppers are designed to sustain greatest stress in a vertical and lingual direction while the lowers best support a vertical and buccal load.[57] If there is a normal physiological bite, without interference of the working excursion of the mandible by cuspal and inclined plane relationship, this is the direction of the applied stress.

Recognizing the trauma caused by lateral stresses, *Leonard*[58] explains the clinically observed injury on the basis of mechanical interference altering the biological processes. The fibers of the membrane stretch and tear on the tension or "pull side" while they are crushed between root and alveolar bone on the compression side. Severe and consistent lateral pressure leads to a devitalization of both the periodontal membrane and the lamina dura. If this pressure is not great enough or of long enough duration to cause immediate necrosis but does impinge the periodontium, an inflammatory hyperemia is set-up which causes a solution of sections of the lamina dura as well as weakening of the fibers of the membrane which is discernible in radiographs. If this is continued, devitalization finally occurs which paves the way for epithelial penetration along the root surface with loss of attachment. Lateral occlusal stress is actually a subtle labiolingual extraction movement.

The degree of destruction is not only regulated by the load of the stress but also by its duration and the presence or absence of other local etiologic factors. In a comparatively small number of cases amendable to treatment systemic conditions may be a predisposition to the velocity of the disease, as concluded in the earlier pages of this treatise. Another factor subsidizing the amount of periodontal involvement is the hardness of the teeth,[5, 59, 60] for the greater the compensating wear of the teeth under stress, the less the destruction to their supporting structures. Related to this is the abrasiveness of the diet which was discussed earlier, while the whole phenomena of the wearing of the teeth is directly correlated with the continuous passive eruption of the teeth with its stimulation of the periodontal membrane to deposit cementum. Wear of the teeth should be commensurate with the age of the patient and facets in young teeth are distinct markings of traumatic contacts of a sufficient magnitude to result in gross alveolar changes, after considerable duration, in spite of the visible wear of compensation. The early middle aged patient with excessive occlusal stresses and no apparent wearing of the teeth will inevitably be plagued with greater bone loss, however.

Another most important consideration in the determination of the pernicity of pathological changes resulting from occlusal trauma is one of basic mechanics.[59] With the gradual loss of alveolar bone supporting the tooth the lever action of the lateral stress is increased by a repositioning of the fulcrum further apically. Therefore, as the alveolar crest moves apically the same load exerts progressively greater pressure on the periodontal tissues and the periodontal disease concurrently advances progressively faster.

Remembering these tenets it is easier to comprehend the kaleidoscopic clinical picture presented by periodontal disease, as well as better answer the critics of the occlusal trauma theory. All persons with occlusal

stress do not present clinical signs of periodontal disease because of insufficient duration, of the imbalance, compensative wear of the teeth, musculature and masticatory habits, and an absence of any other local or systemic causes. The duration of lateral stress can not be ascertained by the age of the patient for occlusion is dynamic rather than static, and the loss of a tooth or teeth may cause a shifting of the remaining teeth that will change the cuspal and inclined plane relationships completely. The loss of a tooth or teeth or even the presence of caries may cause a complete change in the masticatory habits of the patient. Any interference of cusps and inclined planes is related to the individual chewing stroke of the individual patient rather than to disharmonies in eccentric positions of the mandible.[61]

The fact that periodontitis exists in some mouths that are free of lateral stresses does not assure this eventuality in other mouths, as occlusal trauma is but one causative element in periodontal disease. Some patients with relatively minor alveolar bone loss present marked mobility of the teeth while others with considerably greater bone resorption have little or no loosening. This clinical difference is most often the result of lateral occlusal stress in the former. One mouth may remain in an acute inflammatory state for a long period of time with comparatively little alveolar bone destruction; a second may have extensive resorption with no apparent clinical inflammation; while a third may have an advanced reduction of the alveolar crest with a duration of acute inflammation equal to the first patient. Some local irritation, possibly aided by a systemic condition, with subsequent bacteriological invasion, is responsible for the first clinical picture in a mouth most likely free from appreciable occlusal interferences. Excessive lateral stress, possibly aided by a systemic condition, is responsible for the seemingly second clinical entity in a mouth maintained in a hygienic state. The third case undoubtedly possesses a combination of several etiological factors including lateral occlusal stresses.

A "well cared for" mouth that showed alveolar atrophy commensurate with the patient's age upon annual examination for a number of years, and then abruptly appears to go into a progressive degenerative periodontal disease with marked bone loss over a two or three year period may be explained by the progressive rapidity of periodontoclasia caused chiefly by lateral stress, due to the disclosed "increased lever" phenomena. The author is inclined to believe that many of the so-called cases of periodontosis are periodontal involvement from mainly occlusal trauma after the case has reached the progressive cycle. The fact that the histopathological picture is one of inflammation followed by necrosis suggest an irritative cause rather than of systemically caused degeneration. To suppose that a pathological process of inflammation can present such subtle inflammatory changes and yet be followed by rapid necrosis is not absurd when one considers that single rooted teeth have been extracted in hemophiliacs by the gradual and relatively painless apical advancement of a rubber band along the root surface.[62] Smith[63] treated a case that had been examined by both physicians and dentists in several major cities for the possible cause of a believed progressive degeneration of the alveolar bone around both upper central incisors. After an exhaustive search for systemic cause, a flap operation was finally performed and a radiolucent rubber band, which some dentist had placed around the teeth in an effort to correct a diastema, was found to be the cause of the bone destruction. The histopathology in both these cases is almost identical to that of periodontosis.

CONCLUSION

Although many systemic conditions have been suggested as the etiology of periodontal disease, only a few well defined diseases in their severe forms have actually been proved to alter the periodontal tissues. Among these are true scurvy, acute diabetes mellitus, and acute leukemias. The destruction of the alveolar bone in these cases is a symptom of the general malady rather than a true case of periodontoclasia. A subscorbutic vitamin C deficiency may reduce the resistance of

the periodontal tissue to local irrtation, but alone it is not capable of causing periodontal disease or is any other nutritional deficiency. Puberty, pregnancy, or menopause periodontal disease is a myth, but any of these conditions may predispose the gingival tissue to excessive proliferation in response to a local irritation. Chronic leukemias and other blood dyscrasias do not manifest themselves in periodontal alteration. The gingiva may present an allergic reaction to specific drugs but usually the symptoms are not limited to the gums and this condition is not related to pyorrhea alveolaris. In view of the recent discoveries in psychiatric medicine phychosomatic disease seems to be almost unlimited; however, there is still need for proof that periodontal disease may occur as a result of psychological disturbances.

The histopathology supports the contention that all periodontal disease, including the so-called periodontosis, is an exudative inflammation which may vary in its amount of terminating necrosis, rather than a degenerative condition caused by some systemic imbalance. There is little to support the theory that all periodontal disturbance is related to the qualitative characteristics of the cementum which are in turn regulated by some mysterious intrinsic derangement. Reattachment of the periodontal membrane appears possible and desirable in the bony-type pocket that extends below the crest of the alveolar bone, but it is a misnomer to call the often achieved close adaptation of the epithelium in the broad "gum-type" pocket a reattachment. In the latter a true attachment is not histologically possible. Bacteriological studies indicate that the oral flora may propagate and invade the soft tissues causing secondary infection and quite possibly destruction through focal necrosis, but is not responsible for the initiation of periodontoclasia.

Salivary calculus deposits are one of the most common primary local causes of periodontitis and "serumal" calculus formation leads to deeper destruction when it is formed secondary to some primary irritation of the periodontal or gingival tissues. Improper restoration of the coronal parts of the teeth due to poor operative dentistry often plays a paramount role in the etiology of periodontal distress; and when coupled with abuse of the interproximal tissue through careless technic, truly, insult is added to injury. Trauma from improperly directed occlusal stress emerges as not only the principal causative factor in some cases attributed to unknown metabolic disturbances, but also as a contributing element in many others.

Before selecting any therapeutic procedure, a thorough diagnosis of the etiology of the case under observation should be made. Prognosis is poor indeed if the operator is unable to determine the cause of the disease. The removal of the causation of the disease should be the essential ambition of any periodontal treatment, followed by the use of adjuncts to promote the return of the tissues to a state of health and the administration of prophylactic measures to maintain this status. In the "vertical" pocket, every effort should be made to reestablish attachment, while functional adaptation is evidently more desirable than a complete resection of the gums in the horizontal pocket.

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(Concluded on page 196)

Abstracts of Current Literature

49—Swartz, J. H.: **Moniliasis of Mouth, Larynx, One Finger and Vagina.** *Archives of Derm. and Syphil.* **58**:647, 1948.

An 11 year old white girl, E. F., is presented with mucocutaneous lesions of four years' duration. The first complaint was soreness of the mouth and white patches were discovered on the buccal mucosa. Shortly afterward swelling and tenderness developed in the terminal phalanx of the left middle finger. At some unknown later date, the patient experienced vaginal pruritus with lesions similar to those in her mouth. There have been partial remission and exacerbations of symptoms at irregular intervals.

This little girl's mouth exhibits lesions involving the buccal mucosa and various portions of the tongue. These consist of sharply defined, whitish, firmly adherent membranes. There is fissuring at both oral commissures, with a slightly glazed appearance. The labia minora exhibit whitish patches similar to those in the mouth. The terminal phalanx of the left middle finger is slightly swollen. There is thickening of the finger nail, with distortion and opacity of the nail plate.

This patient spent several months in the Massachusetts General Hospital. Laryngoscopy showed the same whitish membranous patches in the larynx. Microscopic examination of scrapings from the mouth, vagina and finger nail revealed sporelike bodies suggestive of monilial organisms. The same material planted on Sabouraud's mediums produced colonies identified as Monilia albicans. Results of roentgenologic examination of the chest were normal. An intradermal test with oidiomycin (1:1,000) resulted in a positive reaction (1 plus). Treatment consisted of general supportive measures, such as high vitamin—high caloric diet and several small transfusions of whole blood. Daily inhalations of ethyl iodide were administered for six weeks. Local applications included streptomycin, 20 per cent sodium caprylate solution, proprionic acid-sodium proprionate solution and quartenary ammonium compound. The progress of the disease has continued uninfluenced by any of this treatment.—*H.M.G.*

50—Wise, F.: **Pachyonychia Congenita with Keratoderma of Palms and Soles and Leukoplakia Oris.** *Archives of Derm. and Syphil.* **58**:608, 1948.

S. H., a woman aged 40, registered at the Skin and Cancer Unit on Jan. 23, 1947, presenting various cutaneous lesions. She has a son 16 who has similar lesions and who is being presented simultaneously. A daughter aged 19 is said to be normal, and there is no history of a similar disturbance in other members of the immediate family or in distant relatives. From the age of 1 year until she was 7, the patient every year had "skin sores" all over the body; these became blistered and later scabby and crusty. They would last all summer, so that she was obliged to stay in the hospital. At the age of 22 she had an infection of the mouth, and 25 teeth were extracted at one time. Her hands and feet are always cold. Menstruation has been regular. She has been having migraine headaches since the age of 24. Her general health has been good except for difficulty in walking because of the callouses. She is intelligent and alert.

A week or two after birth the nails of the fingers and toes became discolored and thickened, reaching their height within a month or two. They have changed little since that time. A slight injury causes an infection and the entire nail is cast off. The lesions on the feet first appeared at the age of 18 months, when she began to walk, and became more pronounced and progressively thicker as she grew older, so that at times her mother was obliged to pare them off. They are painful on walking. In 1933 a skin graft was performed on the left heel, with poor results. She has a continuous tired feeling.

At the points of pressure on the soles, heels and toes, symmetrically distributed, are hard, thick, painful, hyperkeratotic, lemon-colored growths, tender to touch. On the right palm at the base of the metacarpals and the inter-

phalangeal joints are similar growths. All the toe nails are long, thickened, curved, discolored and tapering off toward the end, with the nail of the left ring finger partially broken off. On the right buccal mucosa are small, match head-sized, leukoplakic patches. The entire dorsum of the tongue is covered with whitish, adherent patches and with leukoplakic areas which are particularly noticeable on the sides. The scalp is covered with dry, scaly, diffuse, seborrheic patches. There are two hairy, pea-sized, pigmented moles on the chin. The hands and feet are cool to touch. The integument is dry.

The Mazzini reaction was negative. The urine was normal. The hemogram revealed a normocytic anemia, with 3,710,000 erythrocytes, a hemoglobin contect of 12.2 Gm. per hundred cubic millimeters (73 per cent). The color index was 98. The leukocyte count showed 8,000 cells per cubic millimeter, with 50 polymorphonuclear neutrophils, 41 lymphocytes and 5 monocytes.

An ophthalmologic examination including the eyegrounds and a slit lamp examination of the cornea revealed nothing abnormal.

The total carotinoid content of the blood was 170 units per hundred cubic centimeters (normal, 50 to 100). The vitamin A content was normal.

Vitamin A, 75,000 units daily for a period of six weeks, was taken until three weeks ago. Since February 19 the patient was advised to continue taking 50,000 units of vitamin A and 10 mg. of vitamin E three times a day and ¼ grain (0.015 Gm.) of thyroid extract. Salicylic acid salve, 20% was prescribed, to be applied to the callouses three times a day.—*H.M.G.*

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51 Newman, B. A.: **Thrush, Persisting for Fifteen Years.** *Archives of Derm. and Syphil.* **58**:588, 1948.

H. S., white girl aged 15, presents an illness which dates back to the age of 6 months, when an acute inflammation of the mouth with severe edema developed. At this time, she was a patient in the Los Angeles Children's Hospital and the diagnosis of thrush was made. Since then she has had persistent lesions of the oral mucosa. Treatment had been continuous until three or four years ago and included methylrosaniline chloride given locally, curettage and copper sulfate administered locally; vitamins of all types, given orally and parenterally and liver extract. She was presented at this society five years ago with the same condition.

Physical examination shows the entire oral mucous membrane except the dorsal surface of the tongue and hard palate to be covered with a thick milky white membrane, flaky and patchy in appearance so that it resembles exfoliating mucosa. Some of these membranous patches are easily detached, and others are fairly firmly attached to the underlying tissue, so that on forceful separation, there is profuse bleeding. The general physical condition of the patient is good; the blood cell count and urine were normal; repeated cultures on Sabourand's medium gave prompt growth of Monilia albicans.

DISCUSSION

I believe that there should be some evidence of tissue invasion before a diagnosis of "thrush" is warranted. This is usually manifested by tenderness, ulceration, pain and the presence of bleeding points when a portion of the monilial "membrane" is removed. This patient has had none of these manifestations at any time, and I believe that the proper diagnosis is saprophytic moniliasis. It is possible to obtain cultures positive for Monilia albicans from the mouth, intestinal tract and genitalia of many apparently normal persons, and many probably harbor the organism for years. The dividing line between saprophytic and pathogenic monilial infestation is difficult to draw. The factors which cause the development of the disease in the presence of this organism in some persons while not in others are not well known. It seems probable that deficiencies in some of the vitamin B factors may be partially responsible, notably riboflavin and adenylic acid. I believe that local chemotherapy would not be successful in any large number of cases such as this

one and such attempts might cause breaks in the continuity of the membranes sufficient to allow pathogenic invasion.

I would like to suggest the possibility that this is a nevoid condition, in view of the fact it has been present all the child's life. If it were moniliasis of the mouth, I think that there would be some reddened eroded areas in the mouth and involvement of the tongue. About the culturing of Monilia, one can find that in the normal mouth. I wonder whether Sabouraud's agar or cornmeal agar was used. Sabouraud's agar would reveal only a monilial organism. On cornmeal agar one could see the usual waxy growth and on culture mount find terminal chlamydospores if the organism present is M. albicans.

This patient was completely studied in the past, and at the Mayo Clinic the organism was found in the lesions.

I would call it a leukoplakia-like verrucous lesion of the mouth—possibly the white sponge nevus of the mucosa.

This is an exceedingly interesting condition. I took a tongue blade and scraped the inside of the cheek. The mucous membrane was beefy red and not normal. I could not believe that this was a saprophytic invasion. I agree with the diagnosis of the presenter.

I believe that this is a saprophytic process involving the entire intestinal tract. The organism can probably be cultured from stool specimens as well as from vaginal smears. It is entirely possible that cultural studies will prove the presence of M. albicans of potential pathogenicity. The fact remains that no disease has been demonstrated. I was one of the early examiners, and I could see no erythema even after removing a bit of membrane. The patient has never complained of discomfort; she said that she is conscious of the condition only by constantly chewing small sections of the membrane from her cheek.—*H.M.G.*

52—Bernier, J. L.: **Oral Diseases and Tumors Complicating Periodontal Diagnosis and Treatment.** *J.A.D.A.* **39**:25, 1949.

An understanding of anatomy, physiology and pathology is essential to the diagnosis and treatment of periodontal disorders. Although most patients seeking periodontal treatment exhibit the obvious clinical manifestations of periodontal disease, frequently they have primary or secondary complications in the form of specific manifestations of systemic disease, or perhaps a tumor which is not readily demonstrable by clinical means. The periodontist must be familiar with the diseases of the mouth so that he can accurately interpret conditions that may confront him in his practice. In a sense the periodontist is a practitioner in oral medicine and as such has the responsibility for diagnosing disorders in the mouth as a requisite to proper care of the patient. To this end he must spend some time in the study of the effect of systemic conditions on periodontal diagnosis and treatment. The time spent by the periodontist in such an endeavor will provide ample reward by increasing his ability to interpret the usual clinical pictures and by enabling him to outline and implement complete and satisfactory treatment.

Histologic examination is a valuable means of establishing diagnosis and the result in information often simplifies the problem of therapy.

Such lesions as leukoplakia, lichen planus, keratosis, and the verrucae do not as a rule involve the gingivae; however, their presence in the mouth may present definite problems in periodontal treatment.

Certain gingival manifestations are associated secondarily with inflammation and it is necessary to regard them as distinct from periodontitis because of their specific nature. Examples are gingivitis caused by ingestion of diphenylhydentoin sodium and hormonal gingivitis.

Benign tumors may be seen arising from the periodontium. Among these is the peripheral fibroma which arises from the periodontal membrane. The peripheral giant cell fibroma also arises from the periodontal membrane. Some authors believe that pregnancy and hyperparathyroidism may be of significance in the background of this tumor. All peripheral fibromas associated with periodontal membrane must be removed before periodontal treatment is instituted.

Fibromatosis gingivae (elephantiasis gingivae) is a neoplastic condition making its appearance in early life. Its origin has not been satisfactorily explained although both hereditary and endocrine factors have

been suggested. This condition should be differentiated from gingivitis due to diphenylhydentoin sodium by the taking of a complete medical history. Histologically this lesion is indistinguishable from simple fibroma.

Ossifying fibroma is a slow-growing, benign lesion which is most often encountered in women in middle life. According to some investigators it is a true neoplasm of medullary origin; according to others it is similar to fibrous displasia of bone. Expansive growth may produce considerable deformity and the prognosis is favorable unless adjacent vital structures become involved. There is no constant microscopic picture with the proportions of fibrous tissue and bone varying. With time the bony trabeculae enlarge and the fibrous tissue becomes increasingly collagenous, while vascularity initially prominent diminishes.

Osteitis fibrosa cystica, the generalized decalcification of the skeleton is mainly a manifestation of hyperparathyroidism. It generally affects middle-aged persons and is more common in women than in men. Osteoporosis is demonstrable in roentgenograms, the skull often showing a characteristic mottled effect and the mandible evidence of osteolysis. Diagnosis on the basis of microscopic appearance alone is difficult, because the picture is simply that of osseous repair. Cyst formation is common.

Osteitis deformans (Paget's disease) is relatively rare and may affect only the bones of the skull and mandible. It is more common in women than in men and is usually seen late in life. Changes often occur in the jaws sooner than in other parts of the skeleton and are evidenced by loosening of the teeth. X-rays reveal fungating, superficial deposits of irregular bone which produce enlargement of the part involved. Histologically the bone reveals a mosaic pattern which is the result of overactive resorption and deposition of bone. Osteoclasts are very much in evidence but the process of apposition is slightly in advance of the process of resorption. Exaggerated deposits of secondary dentin have been reported in the pulps of teeth involved in this process.

All these bone lesions may seriously complicate the simple periodontal treatment. Periodontal treatment must usually be modified because of the lesions, but whatever can be done to preserve the masticating mechanism will add to the comfort of the patient.

Eosinophilic granuloma first described in 1940 has been said by many to represent an early phase of Hand-Schuller-Christian disease. It occurs in children and young persons producing tumor-like destructive lesions of the bone. Roentgenograms show that the lesion is osteolytic frequently involving a series of bones. When the jaws are affected, the teeth may become irregular and occasionally may be exfoliated. Cases have been reported in which the initial lesion resembled a periapical abscess and extended rapidly after extraction of the tooth. Histologically the lesion is characterized by masses of lipoid-filled macrophages and the eosinophilic leukocytes enmeshed in a delicate reticular network.

The buccal and lingual gingivae are not as frequently the sites of squamous cell carcinoma as are the mucous membranes in other areas of the mouth. A favorite site of such lesions is the angle of the body of the ramus where ulceration as a result of secondary infection is common. Carcinoma of the buccal mucous membrane is usually preceded by long-standing leukoplakia which has become resistant to treatment. Metastasis may be early and extension wide. The tumor ulcerates early, and the expanding rim is raised and indurated by the young subepithelial tumor nodules. Teeth in the path of the lesion may become loosened and exfoliated. Primary carcinoma affecting the bone substance of the maxilla and mandible has been reported. It is possible that it may arise from epithelial elements of the enamel organ or Hertwig's sheath. In old, extensive lesions the tumor elements invade the surrounding tissue to such a degree that local surgical measures are of little value. Tumors affecting the oral mucosa tend to ulcerate early and widely because of the lack of a well-developed stratum corneum and because of the moisture and warmth in the mouth which favor infection.

CONCLUSION

While most periodontal diseases are essentially inflammatory manifestations, it is obvious that other specific and more serious diseases may occur in the oral cavity simultaneously. In some instances they may appear to be associated etiologically with the periodontal manifestations, although this is not necessarily the case. From the standpoint of diagnosis it is essential that the true nature of any oral disease be thoroughly investigated, regardless of whether or not periodontal treatment is to be attempted. Often teeth may be retained even in the presence of serious disorders. Under such conditions it is usually necessary to modify the periodontal treatment; indeed, it may often be possible or advisable to combine such treatment with other surgical procedures, with emphasis on the more serious disease.

It is important that all diagnostic procedures be utilized, particularly for lesions affecting the mandible or maxilla. The extensive treatment required for complicated disorders often can best be carried out by the periodontist.—*H.M.G.*

53—Lite, T.: **Mouth Breathing: A Contributing Factor in the Etiology of Gingival Irritation.** *J. Dental Med.* **4**:13, 1949.

Mouth breathing produces a deleterious effect upon the mucous membranes and gingival tissues of the mouth. The degree to which this abnormal method of respiration is present determines the amount of gingival inflammation. A decided gingival irritation occurs regardless of whether mouth breathing is indulged in continuously or part of the time. Should this type of abnormal respiration continue, there will result a progressive increase in the pathosis of the tissue.

Any gingival disturbances, if left untreated, may predispose to periodontal disease at a future date. The paper discusses the causes, effects and treatment of mouth breathing in relation to tissue reaction.

Mouth breathing contributes to gingival irritation. This is induced by decreased gingival stimulation and dehydration of the tissues. The cause of oral respiration is comprised predominantly, of nasal obstruction, habit, malocclusion, systemic disturbance, and psychosomatic factors.

Treatment consists of two phases: (1) elimination of the etiologic factors, which may necessitate consultation with the other specialties in the practice of medicine; (2) local treatment, which is dependent upon the elimination of all the sources of local irritation, and providing protection and increased stimulation.

Local treatment consists of:
(1) Toothbrushing and massage
(2) Scaling
(3) Curettage
(4) Equilibration of occlusion

Protection against dehydration of the tissues induced by the continual flow of air is supplied by the application of protective covering. Petroleum jelly has been used successfully.

Etiological Factors in Mouth Breathing

I. Nasal obstruction

A. Irritation or thickening of the mucous membrane of the nose.
   a. Acute rhinitis (head cold)
   b. Chronic catarrhal rhinitis
   c. Chronic hypertrophic rhinitis
   d. Atrophic rhinitis
   e. Vasomotor rhinitis

B. Bone and tissue changes (anatomical) due to pathology
   a. deviation of nasal septum
      1. Heredity
      2. Trauma
   b. Neoplastic rhinitis
      1. Benign
      2. Malignant
   c. Hyperplasia of Adenoid tissue (Pharyngeal vault)
   d. Syphilis of nose
   e. Foreign bodies in the nasal cavities

C. Mouth habits, keeping the lips and teeth apart.
   a. Thumbsucking
   b. Lip biting
   c. Tongue thrusting
   d. Finger-nail biting

D. Abnormal function of the musculature
   a. Direct traumatic injury
      1. Period of development
      2. Temporary
   b. Hypotonicity of the musculature due to organic disease
      1. Hyperthyroidism
      2. Hypogonadism
      3. Nerve destruction
   c. Macroglossia
      1. Hyperpituitary
      2. Hypothyroidism
   d. Hereditary underdevelopment of the musculature

E. Abnormal development of the musculature and the bones of the face
   1. Endocrine dysfunction
      a. Pituitary deficiency
      b. Hypothyroidism
      c. Thymic persistence

F. Malocclusion
   Class II: Division I (Protruding upper teeth, narrow arch, and high palate)

G. Psychosomatic factors.

—*H.M.G.*

54—Brown, W. E.: **Oral Manifestations Produced by Early Irradiation: Report of a Case.** *J.A.D.A.* **38**:754, 1949.

This is the report of a case of a boy who underwent massive roentgen therapy at the age of two years because of a malignant lesion of the mouth. The treatment destroyed the lesion, but also interfered with the growth of much normal tissue.

At two years of age the two lower left molars became loosened and were extracted. The sockets did not heal and the soft tissue in that region ulcerated. Six months later after treatment with various medicaments he

was hospitalized for thorough study. X-ray examination revealed an irregular bony defect in the molar region of the left side of the mandible. Biopsy showed monocytic lymphoblastoma. The patient was treated with radiation, 2500 r and dismissed. The prognosis was extremely unfavorable.

Three months later the patient exhibited cervical adenopathy and a cystic lesion in the region of the symphasis of the mandible with metastasis to the left supra-orbital ridge. Patient was again treated by irradiation. The patient was seen again at four years when he received radiation, at four years and two months and five years and four months, at which time there was no obvious intraoral lesion. There was complete healing at the site of the destructive lesion in the left frontal region and the lesion in the mandible was no longer active. The body of the mandible had undergone almost complete destruction showing marked recession of the bone. The patient was completely edentulous. During this period a newly erupted tooth on the left side of the mandible and a lower anterior tooth was removed because of loosening.

The patient returned for treatment at the age of eleven years and eight months to improve both cosmetics and function. On entrance to the clinic the boy presented a rather unusual physical appearance. He weighed but 50 pounds and was 49 inches tall, which was small for his chronologic age. His face was described by some observers as that of an "old man." The mandible was markedly underdeveloped in relation to the rest of the face. The mouth was extremely small. Intraoral examination revealed four teeth in the maxilla: two fully erupted, widely spaced central incisors with brownish stained, hypoplastic enamel, and two well developed but only partially erupted cuspids, which had normal-appearing enamel. The maxilla was short and the vault low. The mandible was completely edentulous. Owing to the unusual destruction of bone, there was almost no ridge, and in the anterior region the labial movable tissue was attached almost at the crest of the ridge.

Roentgen examination showed several unerupted teeth. There were two bicuspid-like teeth in the left side of the maxilla, one molar-like tooth in the area of the right tuberosity and, on either side of the mandible in the region of the angle, a dwarfed molar crown, perhaps the remnant of a third molar. Right and left lateral roentgenograms of the jaw showed extreme atrophy and notching of the body of the mandible. The inferior dental canal on either side appeared to penetrate to the surface of the bone at the crest of the alveolar ridge. The roots of the unerupted central incisors and cuspids were extremely short. This condition played an important part in the final treatment.

Since an accurate dental history was not kept before the patient's admittance to the clinic, it is interesting to postulate the reasons for absence of the teeth. Probably the portions of the teeth already calcified at the time of irradiation were not affected to any appreciable degree. However, those portions of the developing teeth not imbedded in calcium salts undoubtedly were affected extensively by the roentgen therapy. This conclusion receives support from the stunting of the roots of the remaining teeth and the presence of dwarfed molar-like crowns, near the angles of the mandible. It may be that many of the teeth in the mandible developed, erupted and then became loosened and required extraction because bony support was lacking. It may be possible that the marked retardation of the general bodily growth of the boy was due to extensive irradiation of the pituitary gland. At any rate, one may conclude that heavy doses of roentgen radiation were extremely detrimental to the normal development of the patient's face, jaws and teeth, and possibly to his entire body, although the treatment saved his life.

—*H.M.G.*

55—Morris, P. P., and Reed, R. R.: **Halitosis, Variations in Mouth and Total Breath Odor Intensity Resulting from Prophylaxis and Antisepsis.** *J. Dent. Res.* **28**:324, 1949.

An experiment was undertaken to secure

general information concerning, amongst other things, the intensity of odors commonly appearing on the breath to determine the relative proportions originating in the mouth and that carried by expired air. A study of certain habits and mouth conditions that can lead to relatively intense breath odors was made. Also certain observations were made concerning the putrefaction rate of saliva in vitro and in vivo. The clinical groups were chosen from individuals of both sexes with ages ranging from 18 to 60 years and from an economic status which ranged from unskilled workers to executives with a special group being comprised of university students.

In screening, all subjects exhibiting serious dental or undetermined systemic involvement were discarded. The evaluation of mouth odors was made by the use of the Osmoscope with a potential of odor (p0) of three or above considered describable as halitosis. Sixty-five percent of the subjects had frequent odor levels of 3 or above, (p0 above 3).

Saliva was found to putrefy rapidly on incubation giving rise to various objectionable odors. It was suggested that this along with decomposing protein matter, tobacco odors, as well as odors from lungs contributed to the odor intensity of expired air. The odors of expired air were broken down to those found in the mouth plus those originating in the lungs. There are odors that are of systemic origin which are resistant to any efforts to erradicate them by the use of antiseptic rinses, dentifrices, etc. Smokers appear to have a low mouth and breath odor other than that of tobacco.

Mouth and breath odors were reduced to unobjectionable levels by the use of antiseptic mouth rinse, dental prophylaxis, dentifrice, and tongue prophylaxis with a mouth rinse being the most effective. Water rinses were ineffective in reducing mouth and breath odors.—*H.M.G.*

55—Shpuntoff, H. and Rosebury, T.: **Infectivity of Fuso-Spirochetal Exudates for Guinea Pigs, Hamsters, Mice and Chick Embryos by Several Routes of Inoculation.** *J. Dent. Res.* **28**:7, 1949.

Fusospirochetal exudates were derived from a case of chronic suppurative periodontoclasia, passed through guinea pigs at least 14 times, and stored when necessary by freezing in CO_2 ice. Such frozen exudates, when thawed rapidly after storage for as long as 13 months, were found to contain typical microorganisms with apparently unimpaired motility, and to yield characteristic infection with dosages similar to those required with fresh exudates.

The following hosts were found susceptible to infection by the routes noted: guinea pig, subcutaneous or intraperitoneal; hamster, subcutaneous; and mouse, intraperitoneal or intracerebral.

Infectivity, as determined by successive dilution, is uniformly low, about the equivalent of 0.01 ml. of undiluted exudate being required for a minimal response. There was considerable variation in individual response, for example, the equivalent of 0.1 or 0.2 ml. of undiluted exudate was frequently a fatal dose, but in some instances failed to infect. No marked differences in infectivity were correlated with species or route of inoculation among the adult hosts.

Differences in pattern of response among the hosts and routes employed tend to favor subcutaneous inoculation in the guinea pig both for passage and for titration of infectivity.

The intracerebral route in mice, and all routes of inoculation tested in chick embryos, seemed less suitable than other methods, apparently because of excessive sensitivity to infection with microorganisms other than spirochetes. Typical fusospirochetal infection occurred in only 2 of 54 intracerebrally injected mice, and could be obtained irregularly in chick embryos only by injecting agar with the inoculum into the amniotic sac. Such infection in chick embryos could not be passed to fresh embryos.

Mice that survived intracerebral inocula-

tion seemed as susceptible as previously uninoculated mice to subsequent intraperitoneal infection.

Of the routes of inoculation and the animal hosts tested with fusospirochetal exudates, the subcutaneous method in the guinea pig appears to be most useful for maintaining the exudate by passage, and no less satisfactory than the other methods for titration. Relatively large volumes of exudate were most often obtained in the subcutaneously inoculated guinea pig; and, although both hamster and mouse seem slightly more susceptible, the differences are small and do not offset the advantage of a more characteristic and more easily recognized picture of fusospirochetal infection in the guinea pig.

57—Wolfson, S. A.: **Black Hairy Tongue Associated with Penicillin Therapy.** *J.A.M.A.* **140**:1206, 1949.

Local reactions from the use of oral penicillin (glossitis, stomatitis and versicular cheilitis) have frequently been described. However, another type of reaction has been reported in the foreign literature but not in the American journals. The condition itself, black hairy tongue, has been known for many years, but its association with penicillin therapy is of recent note. Following are the reports of four cases:

Case I—In January, 1948 a white woman aged 37 had been using penicillin lozenges for a sore throat. Discoloration of the tongue was observed a few days later. She was examined on the eighth day at which time the tongue presented a velvety appearance and was brownish black on its medial and posterior portions. The filiform papillae were hypertrophied and bent like the nap of wet, heavy velvet when stroked with a tongue blade. The discoloration gradually cleared, and there has been no recurrence. The significance of the occurrence was not realized at the time.

Case II—A white man aged 59, who had been admitted repeatedly to the Wadsworth General Hospital for treatment of bronchitis, was admitted again on July 12, 1948 for treatment of the same condition. Penicillin had not been used previously, but aerosol penicillin was administered on this admission. A black hairy condition of the tongue developed. No note was made of the date on which the discoloration first appeared. Penicillin therapy was discontinued. The tongue was treated with 50 per cent trichloracetic acid, and the abnormality cleared completely. The patient had subsequent admissions at which time the tongue was normal.

Case III—A white man aged 59 was admitted to the Wadsworth General Hospital on Aug. 12, 1948 for treatment of bronchitis and pneumonitis. The pneumonia cleared following the intramuscular injection of 50,000 units of penicillin every three hours for eight days (Aug. 12 to 20, 1948). However, the bronchitis persisted and aerosol penicillin, 50,000 units twice a day was administered from September 15 to 30. No note was made of the time when the discoloration first appeared, but typical black tongue was seen on September 23, after eight days of therapy. The color was a greenish black and involved the back, medial furrow and pads but not the tip or sides of the tongue. Filaments teased from the tongue resembled, morphologically, the filiform papillae of black hairy tongue. In addition, Candida was cultured from the tongue scrapings. The tongue returned to normal when penicillin therapy was discontinued. The patient was last examined on April 30, 1949 at which time the tongue was still normal.

Case IV—A white woman aged 34 had used penicillin torches for five days, omitted them for four days and resumed taking them for two more days. The tongue showed discoloration after the first five days and became progressively darker. She was seen on April 19, 1949, five days after all therapy had been discontinued. The filiform papillae of the posterior, medial and pad portions of the tongue were hypertrophied and had a heavy dark brown color and plush-like appearance. Warm, saline mouth rinses with gentle scraping of the tongue were recommended. The patient was seen periodically, and the tongue was normal again by May 14, 1949.

Four cases of black hairy tongue associated with penicillin therapy are reported. The opinions of previous observers regarding the possible modes of action of the penicillin are discussed; however, the precise mechanism of its action is yet to be demonstrated.—*H.M.G.*

The most likely theory seems to be that of P. D. Bedford (*British M. J.*, July 13, 1946) that the discoloration is due to a fungus or to a chromogenic bacterial growth, a sequel to the inhibitory action of the penicillin on the usual flora, which later ordinarily inhibit these other organisms. However, Cross failed to recover fungi or bacteria in the cases he examined. As an illustration of the prevalence of this reaction to penicillin, Cross

(*British* M. *J.*, Jan. 29, 1949) noted discoloration in twenty percent of a group he observed for that particular effect, and Wolfson thinks that these figures are not too high. The precise mechanism of this action of penicillin is yet to be determined. There is no specific therapy for the coloration but the condition resolves fairly rapidly when therapy is discontinued. Adjunctive treatment with mouth washes are of doubtful value and strong oxidizing agents of an irritating nature are to be avoided.—*C.R.*

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58 (a)—Shaw, Clarence. **Black Tongue** (Correspondence). *J.A.M.A.* **141(2)**:148. Sept. 10, 1949.

With reference to Wolfson's article in J.A.M.A. 140:1206, Aug. 3, 1949, Shaw states that he described the condition in Conn's "Current Therapy," pointing out that black tongue may be precipitated by penicillin orally, which seems to upset the biologic balance of organisms in the mouth allowing chromagenic bacteria or fungi to multiply. Also in "Diseases of the Skin" (Sutton and Sutton), observations of Burgheim are reported. The latter noted black tongue following sulfanilamide treatment of patient for gonorrhea which disappeared when sulfa therapy was discontinued.—C. R.

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58 (b)—Wolfson, Samuel A., in *J.A.M.A.* **141(11)**:794. Nov. 12, 1949.

Commenting on letter of Clarence Shaw (see above), Wolfson commends and confirms reference to effects of sulfonamides as well as penicillin as causes of black tongue. Still others have reported as causative repeated applications of tinctures containing vegetable coloring matter (in which the principal offending agent seemed to be tinctura Krameria), and also streptomycin (corrected later to read "Chloromycetin"). The list will probably grow as new therapeutic agents are introduced.—C. R.

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58 (c)—van Klaveren (Bombay, India). **Black Hairy Tongue.** *J.A.M.A.* **141(18)**:1319.

Commenting on Wolfson's article in J.A.M.A. for Aug. 13, 1949, Dr. van Klaveren calls attention to observations of Doxiades and Tiliados (Swiss) who noted condition in avitaminosis, same being complicated by other diseases duodenal ulcer, paresis, pernicious anemia. Riboflavin treatment precipitated the tongue condition and treatment with B_1 plus nicotinamide or B complex cured the symptoms. The question comes to van Klaveren's mind—could penicillin therapy increase the requirements of the organism for Vitamin B complex?—C. R.

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58 (d)—Overman, Wm. E. **Penicillin and Black Hairy Tongue.** Report of a Case. *J.A.M.A.* **141(18)**:1319. Dec. 31, 1949.

Overman reports case of Black Hairy Tongue apparently due to medication with penicillin troches. Patient had continued use of the 50,000 unit tablets beyond the amount and period prescribed, and the typical black hairy tongue which developed cleared gradually but steadily on discontinuance of the penicillin with no other treatment.—C. R.

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58 (e)—Ayre, W. Burton, Robt. Favreau and Ernest J. Ayre. **Black Tongue following Penicillin.** *J.A.M.A.* **141(8)**:548 Oct. 22, 1949.

These 3 men at McGill U. in Montreal having noted that black hairy tongue followed use of penicillin, further observed that the dark velvety tufts which form in a few days subsequently slough off if same medication is continued. They now administer penicillin for the purpose of promoting the desquamation of the long black filaments of lingua nigra from other causes. Two cases in point are described. A bald slightly sensitive tongue results, similar to those seen in certain B deficiencies. In one case however patient was having B vitamin therapy, parenterally in heavy doses when desquamation occurred. "Black Tongue" is actually the elongation of the filiform papillae which are ordinarily short cornified tufts, and their desquamation keeps pace with rate of formation and they remain short. Evidently penicillin

either increases in some way the rate of cornification, or alters that which is produced to make it more cohesive. But the desquamation produced by continued use of penicillin is evidently quite a different mechanism. Experiments have shown that penicillin also increased cornification of vaginal epithelium. These studies, so far, seem to suggest that penicillin may directly affect enzymes of the cells involved in the cornification process.—C. R.

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58 (f)—Query: **Black Tongue.** *J.A.M.A.* **141(9)**:634. Oct. 29, 1949.

There is no new or specific treatment for this "lingua villosa nigra," called also Black hairy tongue or filacosa chromatica. This condition is a hypertrophic elongation of the filiform papillae. It is somewhat similar to skin keratosis. Patient should be assured that it is harmless. The cause is uncertain. The various yeasts and fungi which may be present are secondary nor is there any probability that dentures or dissimilar fillings are factors in the etiology. The purpose of therapy is to clear away the growth by scraping or brushing, though it may recur. One per cent aqueous solution acriviolet, 3% hydrogen peroxide or methylrosaniline chloride (or a mixture of the two) may be applied recurrently, but no NaCl nor any boron mouth wash since they are sometimes considered causative. Reference is made to Wolfson's article (J.A.M.A. 140:1206) concerning oral penicillin as a cause. Obviously, this medication should be discontinued.—C. R.

59—Glickman, I., Turesky, S., and Hill, R.: **Determination of Oxygen Consumption in Normal and Inflamed Human Gingiva Using the Warburg Manometric Technic.** *J. Dent. Res.* **28**:83, 1949.

The findings indicate that the oxygen consumption of human gingivae is measurable by the Warburg manometric technique. Although the number of cases does not lend itself to full statistical analysis, it appears that in gingival disease variations in oxygen consumption may be related to microscopical pathological changes.

Because all the operations in this experiment were performed under novocaine anesthesia, the effect of these substances on oxygen consumption must be considered. The literature indicates that both novocaine and adrenalin as contained in this anesthetic depress oxygen consumption. Some qualifications of the Qo 2 (oxygen consumption) levels for the human gingiva obtained in this study is therefore indicated; however, the exact degree to which oxygen consumption of gingival tissue is modified by novocaine anesthesia is yet to be determined. The results of the experiment were as follows:

1. The average oxygen consumption for normal gingivae was 1.6—.37.

2. Oxygen consumption was essentially unaltered in gingivae with slight inflammatory and/or proliferative changes.

3. There was a tendency towards increased oxygen consumption in gingivae with marked chronic inflammation and proliferation of connective tissue and epithelium.

4. There was a tendency towards decreased oxygen consumption in gingivae which were essentially degenerated and necrotic and contained few if any viable tissue elements.

The tendency towards increased oxygen consumption in marked chronic marginal gingivitis is apparently associated with an infiltration of inflammatory cells the formation of new blood vessels and connective tissue fibers and the proliferation of connective tissue and epithelium.

61—Query: **Treatment for Chronic Moniliasis.** *J.A.M.A.* **140(11)**:995 (July 16) 1949.

There is no specific treatment for this fungus infection which in humans is usually found in locus minoris resistentiae. In the mouth it occurs as thrush. The association with diabetes should be ruled out; if infection is purely oral, local sites including gingival crevices should be cared for and the tongue scraped to rid the surface of all filiform papillae. Condition has sometimes been attributed to vitamin B deficiency. B com-

plex concentrate by injection followed by oral doses, and diet correction are advised, and a bland alkaline mouthwash several times a day. Weak gentian violet or acriviolet are usually effective. For severe resistant cases, Roentgen therapy may be used; also autogenous rabbit serum vaccine has been reported successful for severe cases with positive skin tests, a daily subcutaneous hyposensitizing technique being used.—*C.R.*

62—Query: **Sweet Taste Sensation.** *J.A.M.A.* **140(17)**:1377.

A middle aged man in good physical condition has extremely disagreeable sweetish taste in mouth constantly. Possible causes are infection in respiratory tract or mouth, a denture or some mouth wash, although tastes from these causes usually are not "sweet" nor "unbearable." Local causes being ruled out a neuro-psychiatrist should be consulted. Hysteria can produce "constant intense taste perversion." A history of change or stress or illness at time of onset would be significant in etiology.—*C.R.*

63—Query: **Oligosialia.** *J.A.M.A.* **140(10)**:129 (July 2) 1949.

Woman of 74 complains of xerostomia, and what saliva she has is thick and mucoid. She had purulent bilateral parotiditis which cleared under penicillin treatment but left above condition. Usual home remedies, gargles and logenzes have been tried as well as autogenous vaccine, pilocarpine, and potassium iodide. Roentgenograms are negative.

Ans.: Parotid infection plus the age of the patient would seem to be the cause of the trouble. Rule out use of belladonna and astringent mouth washes; the presence of diabetes or chronic nephritis and any evidence of autonomic imbalance. Best drug is neostigmine bromide; 7.5 mg. after each meal produces profuse thin saliva and oral symptoms should subside. The general health should be built up with vitamin concentrates, and oral conditions treated locally as indicated.—*C.R.*

64—Query: **Salivation.** *J.A.M.A.* **140(5)**:504 (June 4) 1949.

Query is in regard to excessive salivation in Parkinsonism. This is a typical symptom in the post-encephalitic form of the disease and usually is rapidly controlled by belladonna and like drugs, adjusting the dosage to the patient's tolerance. An extract of belladonna has been advised, starting with .4 mg. daily and increasing until there is a toxic response, then reducing dose. Some cases tolerate as much as .8 mg. three to four times daily. With improvement in salivary control, muscular function and improvement in swallowing should follow.—*C.R.*

65—Braunstein, Leo E.: **"Lipoma of Tongue."** *J.A.M.A.* **140(2)**:155 (May 14) 1949.

Braunstein reports a case of lipoma of the tongue, an exceptional site for this type of pathology.—*C.R.*

66—Query: **Oral Lesions after Hysterectomy.** *J.A.M.A.* **140(11)**:996 (July 16) 1949.

Symptoms are described in patient's mouth following hysterectomy, the most annoying of which is thick ropy saliva. The mucous membrane of inside of cheeks is indurated, with occasional blisters. She has not responded to hormone, vitamin or liver therapy.

Ans.: Details as to operation, age and general condition of patient are too meager, but there are no oral lesions known to be the direct result of hysterectomy. The type of anaesthetic, dehydration of patient, or the presence of infection might be responsible for some of the symptoms. The induration in cheeks in line with the occlusal surfaces of teeth is probably traumatic. If condition does not clear with restoration to good health and hydration, a few days medication with neostigmine bromide, 7.5 mg. by mouth after each meal, should increase salivary output. No drugs which reduce secretions should be used. Advise a mild soothing mouth wash.

—*C.R.*

67—Query: **Sialorrhea.** *J.A.M.A.* **140(17)**:1374 (Aug. 27) 1949.

Query concerns patient of 70 with severe sialorrhea. He has bronchial asthma and arteriosclerotic heart disease and is taking digitalis.

Ans.: Treatment of salivation depends on cause which in present case may be man's general condition. He may have a basal gangliar degeneration, with stomatitis, smoking and avitaminosis as contributory factors. Drugs, dental difficulties, stomatitis, vitamin B deficiency, reflexes from intestinal and other organic ailments, psychoses and organic disease of the central nervous system are all possible causes. Atropin is the drug indicated and it should be used cautiously, one-fourth mg. twice daily, being alert for possible toxicity. Clean up mouth and prescribe B complex. Irradiation of salivary glands will reduce amount of saliva, but may check flow too radically.—C.R.

68—Query: **Recurrent Herpetiform Lesions.** *J.A.M.A.* **141(4)**:299. Sept. 24, 1949.

A married man of 41 has had recurrent small herpetiform ulcers on cheeks, gums, tongue and palate since 1940, onset of lesions having coincided with the severe bombing of England while he was there in 1940. Only complete freedom from ulcers was during 3 months in England in 1946, although they improved in 1943-44 while patient was receiving weekly injections of calcium glutinate; also temporary improvement resulted from administration of phenobarbital ½ gr. 4 times a day. The condition is decidedly aggravated by emotional stress. There is no history of allergy.

Answer: Etiology of such lesions is complicated although the virus of herpes simplex is primary cause. Susceptibility may be increased by allergy, B deficiency, mild oral trauma, emotional instability and intoxication. The reappearance is due to the characteristic recurrent loss of immunity. In this case the history of psychic trauma is suggestive. An interesting hypothesis as to the manner in which emotions can produce an oral lesion is that mental stresses in some persons give rise to an increased lysozyme content of various secretions. This lysozyme, being mucolytic, reduces the natural protection of the mucosa against the virus. A psychiatrist might help. Barbiturates are suggested, also estrogen ointment applied locally. In many hands repeated smallpox vaccinations up to 8 or 10 have proved best method. Although an oral flare-up may ensue, the ultimate results are usually good.—C. R.

69—Query: **Aphthous Stomatitis.** *J.A.M.A.* **141(5)**:362. Oct. 1, 1949.

Woman of 21 normal otherwise has had aphthous stomatitis almost continuously since childhood being never completely free of it though worse during menstruation. She has been hospitalized for treatment and a great variety of vitamins and antibiotics administered. At present she receives 1 tablet cobefortis (combination of several of the members of B complex with ascorbutic acid) daily; she uses methylrosaniline chloride locally and 1 ampule of bismarsen weekly. Above regimen seems to shorten duration of acute symptoms but has not cured condition.

Answer: The susceptibility to the virus of herpes simplex may be modified by secondary factors, but the lesions are recurrent and not affected by methods used in this cure. Treatment recommended is series of smallpox vaccinations, 8 or 10 at intervals of a week or 10 days, preferably given during remissions—and continued regardless of "takes." Local use of estrogenic ointment has also been suggested. (See other abstracts and articles —July 10, 1940:1004, etc.)—C. R.

70—Query: **Canker Sores and Smallpox Vaccination** *J.A.M.A.* **141(5)**:362. Oct. 1, 1949.

A woman of 30 had severe recurrent oral canker sores. She has had 3 series of treatments in a 12-month period with smallpox vaccinations. Improvement followed treatment, sores becoming fewer, shallower and less painful; but immunity does not last and

patient gets a typical take each time from her first vaccination. Why is immunity so brief in this case and how can it be prolonged?

Answer: Such ulcerations are now thought to be due to the virus of herpes simplex and it is typical of these infections that they do not of themselves produce a lengthy immunity. Usually persons and families with susceptibility of this kind have a low antibody titer. They will often have difficulty getting a "take" from smallpox vaccination. There is no way to prolong immunity but all logical adjuncts should be tried. Allergies should be considered, and emotional instability, and oral hygiene improved. An antihistaminic drug and the new antibiotics might be tried. Possible conditioning factors in the etiology are B complex deficiency, hyperthyroidism, fevers, leukemias, menstruation, mild trauma and constitutional peculiarities.—C. R.

71—Savitt, Leonard E. **Canker Sores.** Case report. *J.A.M.A.* **141(18)**:1330. December 31, 1949.

Savitt considers terms "canker sores," "Aphthous stomatitis" and "herpetic stomatitis" synonymous. He has found the following treatment with smallpox vaccine to be successful in the majority of his refractory cases of recurrent herpes simplex on lips or elsewhere. After the first scratch he waits until reaction from "take" subsides before proceeding with treatment. If no "take" ensues, a mixture of .1 cc. of isotomic NaCl solution with one capillary tube of smallpox vaccine containing "brilliant green," is divided in 4 parts—¼ of this is given intradermally and 3 days later ½. About 10 more injections are made at 3-day intervals preferably though not necessarily in deltoid muscle. If this is not effective 2 capillary tubes of the vaccine with .1 cc. of the saline solution should be given in one dose. If this does not avail, he considers there must be cause other than the herpes virus: Among factors not usually mentioned are—eating walnuts, hard candies, red icings (containing Erythrosin), toothpastes, mouth washes, etc. —C. R.

72—Query: **Taste aberration and sore tongue.** *J.A.M.A.* **141(3)**:237. Sept. 17, 1949.

Advice is asked concerning woman of 60 with complaint of bitter taste and sore tongue for 2 years. There are no objective symptoms.

Answer: If an actual glossitis is meant by "sore tongue" the salty bitter taste is likely a local effect on the taste buds. Glossitis may be due to vitamin deficiency or a low grade infection or a dental fault. Vitamin B treatment is usually indicated, one or several members of the complex according to indications. Careful diagnosis should determine whether anemia is present; and blood tests and gastric acidity tests used to determine the type. Also diet history should be taken; and based on all these findings iron, liver or the newer B_{12} or nicotinic acid should be administered. Severinghaus obtained fine results by giving nicotinic acid for three weeks, 50 mg. a day or more orally. A possible local infection or fillings of different electrical potential, faulty artificial dentures, should be investigated by a dentist. Excessive smoking should be curtailed. If correct therapy for all of the above fails, a psychoneurotic etiology is to be suspected.—C. R.

73—Brown, A. **Glossitis in Pernicious Anemia.** *British Med. Jour.* **1**:704. April 23, 1949. Abst. in *J.A.M.A.* **141(4)**:292. Sept. 24, 1949.

Brown describes 7 patients with pernicious anemia, each of whom had a sore, red, raw-looking tongue. One also had angular stomatitis and vascularization of the cornea. In each case it was possible to control these changes with a single member of the vitamin B complex in pure form. Four cases of glossitis responded to calcium pantothenate and one each to nicotinic acid and folic acid. One case responded first to nicotinic acid, later to riboflavin. The patient with glossitis, angular stomatitis and vascularization of the

cornea responded to riboflavin. It is suggested that the epithelial changes described reflect a breakdown in some metabolic system which may be similar to that responsible for the megaloblastic marrow and the neurologic complications of pernicious anemia.—C. R.

74—Huet, J. A. **Teeth and Endocrine Disorders** (Correspondence from France). *J.A.M.A.* **140(10)**:737. November 5, 1949.

During X-ray examinations of the skull in certain endocrine conditions Huet noted the frequency of morphologic modifications in the teeth and also the coincidence of migration of the teeth especially the upper third molars. In addition he found infectious lesions of dental origin in a large number of cases of infectious thyroiditis and recommends routine X-ray of the skull in all disorders of thyroid or hypophyseal origin. Such cases should be referred to a dental surgeon when there is no response to endocrine therapy.—C. R.

75—Query: **Bruxism.** *J.A.M.A.* **141(16)**:1196. December 17, 1949.

Query concerns a girl 11½ years old who has had an increasing habit of grinding her teeth in her sleep since she was four. She is otherwise normal except for being overweight.

Answer: This habit is in the class of other common childhood sleep disturbances such as nightmares, etc. Among physical factors possibly responsible are hunger, digestive troubles, excessive cold or heat. Stumps of tonsils, enlarge adenoids should be eliminated. These factors having been ruled out, the home environment should be studied for possible causes of worry, anxiety, insecurity, inferiority, tension or over-stimulation of the child's emotions. It is important that all psychological factors be corrected especially in an overactive, nervous child.—C. R.

76—Schaffer, Julius. **Treatment of Vincents Infection.** *J.A.M.A.* **141(2)**:170. Sept. 10, 1949.

Commenting on answer to Query regarding Vincent's Infection in J.A.M.A. 140: 1228 (April 23, 1949), Schaffer says caustics such as silver nitrate and chromic acid are frowned upon since they do permanent damage by destroying tissue. The Vincent's bacterial symbiosis being found in many mouths which do not exhibit the disease. A smear showing these organisms is not reliably pathognomonic. Schaffer finds following treatment uniformly satisfactory. Remove debris immediately from affected area by scaling as thoroughly as patient's tolerance can stand. This is accompanied by frequent spraying with peroxide of hydrogen 50% in warm water. Penicillin troches are prescribed, or in acute conditions 300,000 units are injected intramuscularly. Tobacco, salty, spicy foods and alcohol are prohibited. Patient is given new tooth brush and for home treatment frequent rinsing with warm peroxide solution is advised. All acute symptoms are usually gone in 24 hours when intensive scaling is started and the correction of malocclusion. Rough fillings and other mechanical defects, caries and third molar flaps are eliminated as promptly as possible. Treatment usually requires 3 to 6 visits during which patient is instructed in brushing technique, diet, etc. After clinical symptoms have disappeared tobacco in moderation and normal diet may be resumed.—C. R.

77—Query: **Halitosis.** *J.A.M.A.* **141(4)**:299. Sept. 24, 1949.

Why is it that many people who apparently have no paranasal sinus pathology nor decayed teeth have a sour or decayed breath odor and how can it be treated?

Answer: S. C. Miller lists 14 distinctive odors in halitosis, or foul breath, and treatment depends on discovery of cause which is often obscure, and its elimination. Among causes are, chronically diseased tonsils and adenoids, bronchial infection, chronic nasal catarrh and atrophic rhinitis; various disturbances along the alimentary canal, diverticula, colitis, faulty fat metabolism, gastro-

intestinal allergies. Each requires special treatment. Smoking may be contributory. Mouth conditions are the most likely offenders—dentures, low grade infections of gingivae, enlarged, inflamed papillae and oral bacteria lodged between gums and teeth, especially in the presence of tartar, and food between the teeth, especially meat because protein can become foul in a few hours. Vincent's infection is readily recognized if present. The teeth should be cleaned by the dentist and mouth hygiene maintained assiduously by patient and dentist. "Tone of mucous membrane may be improved by use of dilute mouthwash containing zinc chloride and perhaps preceded by one containing hydrogen peroxide or sodium perborate." References: Crohn, B. B., and R. Drosd—"Halitosis." J.A.M.A. 117:2242. Dec. 27, 1941; Cecil, R. L. Textbook of Medicine; Milton, S. C., Oral Diagnosis and Treatment; Burket, L. W. Oral Medicine.—C. R.

78—Schieve, J. F. and R. W. Rundles. **Response of Lingual Manifestations of Pernicious Anemia to Pteroylglutamic Acid and B_{12}.** *Journal of Lab. and Clin. Med.*, **34**: 439. April 1949. Abs. *J.A.M.A.* **141(3)**:225. September 17, 1949.

Lingual Manifestations of Pernicious Anemia. Schieve and Rundles treated 8 patients with atrophy and inflammation of the lingual mucosa, resulting from pernicious anemia with pteroylglutamic acid. During the first month patients were given 75 to 100 mg. of the drug by mouth and for the next two months 30 to 50 mg. daily. In 2 of these patients treatment with pteroylglutamic acid produced a rapid restoration of normal lingual mucosa. The tongue remained normal, and there was neurologic improvement, in spite of an incomplete, hematologic remission. In 4 other patients pteroylglutamic acid failed to produce an adequate lingual response. Liver extract was more effective in correcting the lingual and other manifestations of the disease. The last 2 patients treated with pteroylglutamic acid had distinct relapses during the third month of treatment, in spite of satisfactory hematologic and neurologic responses. The lingual abnormalities disappeared within five to seven days after the injection of vitamin B_{12}. In 5 patients showing the lingual manifestations of untreated pernicious anemia the injection of vitamin B_{12} in a dose of 0.001 mg. daily to 0.010 mg. in a single dose led to regeneration of lingual papillae in five to seven days. Results suggest that treatment with pteroylglutamic acid has an irregular effect on all manifestations of the pernicious anemia rather than failing consistently in anyone. The cause for individual variability in therapeutic response remains to be determined.—C. R.

79—O'Leary, P. A., Montgomery, H., Brunsting, L. A., and Kierland, R. R.: **A Case for Diagnosis (Tuberculid?).** *Archives of Derm. and Syphil.* **58**:638, 1948.

A. B., an 18 year old Negro girl, has had a generalized pruritic papular and vesicular eruption with involvement of the mouth for the past two years. This has been accompanied with an inflammatory condition of the eyes and by migratory polyarthritis. She gives a history of hospitalization elsewhere from November 1942 to March 1943, because of possible pulmonary tuberculosis.

Physical examination reveals a generalized papular and vesicular eruption of the skin with crusted excoriations, scars and pigmentation, accompanied with erosions on the mucous membrane and an anal fissure; swelling and entropion of the eyelids with injection, bulla formation, superficial ulceration, scarring and cicatricial contraction of the conjunctivas; painless swelling of the right knee, and localized left hydropneumorthorax with fibrous tuberculosis of the apex of the left lung. Results of serologic tests for syphilis were negative. The result of a tuberculin test (purified protein derivative, first strength) was strongly positive. (Microscopic sections were presented.)

DISCUSSION

I thought that there was a possibility that the patient had dermatitis herpetiformis. The lesions were in the areas that ordinarily are involved. The eruption was itchy; many of

the lesions were vesicular, and the microscopic section showed vesicles with a high percentage of eosinophils in the infiltrate.

—*H.M.G.*

80—Swartz, J. H.: **Moniliasis of the Mouth.** *Archives of Derm. and Syphil.* **58**:648, 1948.

An 18 year old white girl, S. B., occupied as a hairdresser, is presented with oral lesions of six months' duration. The patient first noticed enlarged glands in her neck and shortly afterward found white patches in her mouth. These were asymptomatic at first, but spread gradually and there was subsequently some pain on swallowing.

Examination reveals lesions scattered over the palate and anterior tonsillar pillars. They consist of small grayish deposits which create a mottled appearance on the affected areas. This exudate can be easily removed by scraping, revealing a normal-looking mucous membrane. Those on the tonsillar pillars and a few on the soft palate are more whitish.

This patient spent about a month in the Massachusetts General Hospital. It was found that the removal of all exudate was followed by its rapid reappearance in almost identical fashion. Material obtained from the lesions of the mouth grew organisms identified as Monilia albicans when cultured on Sabouraud's mediums. Treatment consisted of the application of Gram's solution and subsequently of 20 per cent sodium caprylate solution, in each instance after removal of exudate. The disease proved refractory to treatment.

DISCUSSION

I would suggest the use of a paint containing 1.0 per cent methylrosaniline chloride and 1.0 per cent formaldehyde, to be applied daily.

There is a solution which I have been evaluating clinically: glycerite of hydrogen peroxide. In the presence of serum and blood cells it liberates oxygen, and because of the surface tension of the glycerine it contains, the liberated oxygen is confined to the solution over prolonged periods of time, for twenty-four to thirty-six hours. I have not used the solution in moniliasis. I do know that certain spore-bearing organisms are killed by oxygenation from this compound. When used as a diluent or for tropical applications, it offers a distinct method of therapy. The solution is now commercially available.—*H.M.G.*

ANNOUNCEMENT

The XIIth Congress of the International Arpa—Madrid, March 13th to 18th, 1951

The XIIth Congress of the International Association pour les Recherches sur les Parodontopathies will take place from 13th. to 15th. March, 1951 in Madrid, the week preceding Palm-Sunday. Tuesday 13th, Wednesday 14th and Thursday 15th of March will be devoted to scientific reports and films concerning Periodontal disease.

The committees of the Spanish Stomatological Society and the Spanish Arpa will organize on Friday 16th a visit to the Escurial and on Saturday 17th a trip to Toledo. On the evening of the 17th there will be the official dinner followed by national festivities.

The participants could therefore have the occasion of visiting Spain during the Easter week which offers a particularly interesting and picturesque opportunity.

Dentists are asked to prepare reports and to keep their time free for the XIIth Congress of the International Arpa (13-18 March, 1951). The President of Arpa is Dr. R. Jaccard, the Vice-President is Professor A. J. Held and the Secretary General is Dr. U. Vauthier.

Announcements

Dr. Robert G. Kesel, Chairman of the Postgraduate Seminar of the American Academy of Periodontology, reports that their annual meeting which was held in Ann Arbor May 25-27, 1950, was very successful. There were over fifty guests in attendance.

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The Southern Academy of Periodontology held their annual meeting on May 21-23, 1950, in Jacksonville, Florida. The program included presentations by both Doctors Leonard and Isadore Hirschfeld, and a series of table clinics and discussions by members of the Academy. The retiring President was Dr. R. P. Taylor, Jr., of Jacksonville, Florida, and the new officers are: President, J. C. Westbrook, Jr., Birmingham, Ala.; President Elect, Roger Turner, Miami; Secretary Treasurer, S. P. Gay, Greensboro, N. C.; Executive Council, R. P. Taylor, Frank Alford, Roger Turner, F. W. Griffin, Richard Chace, J. E. Westbrook, Jr.

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Dr. Hermann Becks, M.D., D.D.S., founder and president of the Annual Seminar for the Study and Practice of Dental Medicine, has announced that the dates scheduled for the Seventh Annual Dental Seminar are October 15-20, 1950.

The Desert Inn at Palm Springs, California, will once more be the gathering spot for this popular meeting of leading dental-medical practitioners from throughout the United States, as well as from abroad.

Total fee of $150 includes registration, lodging and meals for the five days, October 15 to 20. Applications should be mailed to Miss Marion G. Lewis, Executive Secretary, Room 200, Hooper Foundation, University of California Medical Center, San Francisco 22, California.

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Periodontology Courses at Columbia University

Three postgraduate courses in Periodontology are offered at Columbia University, School of Dental and Oral Surgery of the Faculty of Medicine.

1. A full time, two academic year program leading to a Certificate of Training in Periodontology. Correlation of the fundamental sciences to clinical practice is emphasized.

2. One-half day each week from September 20, 1950 to May 2, 1951 in Periodontology.

3. Full time from June 4, 1951 to June 15, 1951 in Periodontology.

For further information write to Dr. Barnet M. Levy, Director, Postgraduate Division, School of Dental and Oral Surgery, Columbia University, 630 West 168th Street, New York 32, New York.

OCCURRENCE OF GINGIVITIS IN CHILDREN

(Continued from page 164)

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ETIOLOGY OF PERIODONTAL DISEASE

(Continued from page 178)

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PLEASE NOTE

Attention is called to the fact that J. Lewis Blass, Ph.G., D.D.S., was a co-author of the article on *"Nutrition and Diet in Periodontic Practice"* published in the April 1950 issue of this JOURNAL. His name was inadvertently omitted from the copy of the paper submitted for publication.

Apologies are also due to Drs. Bell, Rule, Dienstein and Nuckolls because some of the radiographs in their April article *"The Periodontal Lesion,"* did not reproduce very well, in spite of the fact that the JOURNAL OF PERIODONTOLOGY spared no expense in preparing the cuts. Reproduction of radiographs is very difficult; authors are urged to use them only when absolutely necessary.